

**INCIDENCE, RISK FACTORS AND  
COMPLICATIONS OF DYSPHAGIA IN STROKE  
PATIENTS ADMITTED TO MEDICAL WARDS AT  
A TERTIARY HOSPITAL IN SOUTH INDIA**



**A Dissertation submitted in partial fulfillment of  
M.D (General Medicine) branch I Examination of the Tamil Nadu  
Dr. M.G.R. UNIVERSITY, CHENNAI  
to be held in 2016**

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## LIST OF FIGURES

Figure 1: Leading causes of death in the world.....	19
Figure 2: Geographical distribution of incidence of stroke .....	23
Figure 3: Pathophysiology of stroke .....	29
Figure 4: Modified Rankin Score .....	35
Figure 5: Swallowing of a liquid bolus .....	39
Figure 6: Relation between dysphagia and pneumonia .....	41
Figure 7: Sydney swallowing questionnaire .....	44
Figure 8: Video fluoroscopy .....	51
Figure 9: Fibreoptic Endoscopic Evaluation of Swallowing .....	52
Figure 10: Relation between aspiration and pneumonia .....	57
Figure 11: Pathophysiology of stroke associated pneumonia .....	59
Figure 12: STROBE statement .....	71
Figure 13: Risk factor profile of patients .....	72
Figure 14: Location of stroke .....	74

Figure 15: Incidence of dysphagia as assessed by Mann's assessment of swallowing ability .....	75
Figure 16: Severity of dysphagia as assessed by Mann's assessment of swallowing ability .....	75
Figure 17: Speech therapist's diagnosis of dysphagia .....	77
Figure 18: Number of patients with dysphagia using combined assessment with Mann's score, speech therapist's assessment and FEES .....	78
Figure 19: ROC curve assessing performance of Mann's score in diagnosing dysphagia .....	79
Figure 20: Improvement of dysphagia with time .....	87
Figure 21: Incidence rates of pneumonia against the presence or absence of dysphagia .....	88
Figure 22: Relation between severity of dysphagia and pneumonia .....	88
Figure 23: Relation between dysphagia and mortality .....	89
Figure 24: Relation between dysphagia and duration of hospital stay ...	89
Figure 25: Incidence of pneumonia .....	90
Figure 26: Relation between pneumonia and dysphagia.....	91

Figure 27: Relation between mortality and pneumonia.....	96
Figure 28: Relation between pneumonia and duration of hospitalisation..	97
Figure 29: Proportion of patients who came for follow up.....	97
Figure 30: Location of Ryle's tube removal.....	98



## LIST OF TABLES

Table 1: Demographic details.....	71
Table 2: Baseline characteristics.....	74
Table 3: Clinical findings during speech therapist's assessment of swallowing...	76
Table 4: Comparision between clinician's and speech therapists assessment of dsphagia.....	77
Table 5: Assessment of swallowing using Fibreoptic Endoscopic Evaluation of swallowing.....	78
Table 6: Effect of sex and the risk factors of stroke on development of dysphagia .....	80
Table 7: Association of clinical features of stroke with dysphagia.....	81
Table 8: Association of various scoring systems with dysphagia.....	82
Table 9: Clinical swallowing assessment for dysphagia.....	83
Table 10: Swallowing assessment using thin liquids.....	84
Table 11: Swallowing assessment using thick liquids.....	84
Table 12: Fibreoptic Endoscopic Evaluation of Swallowing for dysphagia.....	85

Table 13: Logistic regression for factors predicting dysphagia.....	86
Table 14: Association of sex and risk factors of stroke with pneumonia.....	91
Table 15: Association of clinical features of stroke with pneumonia.....	92
Table 16: Association of age and various scoring systems with pneumonia.....	93
Table 17: Association of clinical findings of swallowing assessment with pneumonia.....	93
Table 18: Association of clinical findings of swallowing assessment using thin liquid with pneumonia.....	94
Table 19: Association of clinical findings of swallowing assessment using thick liquid with pneumonia.....	94
Table 20: Association of findings during FEES with pneumonia.....	95
Table 21: Logistic regression for independent factors associated with pneumonia.....	95

## CONTENTS

S.No.	Content	Page No.
1	Introduction	17
2	Aims of the study	20
3	Objectives of the study	21
4	Review of literature	22
	Global epidemiology of stroke	22
	Epidemiology of stroke in India	23
	Definition of stroke	25
	Ischaemic stroke	27
	Haemorrhagic stroke	31
	Clinical features of stroke	32
	The problem of dysphagia in stroke	36
	Normal swallowing	37
	Dysphagia	39
	Significance of dysphagia	41
	Methods to diagnose dysphagia and aspiration	42
	Role of speech therapist	54
	Pathophysiology of dysphagia	56
	The problem of aspiration pneumonia in stroke	56
	Pathophysiology of aspiration pneumonia	57
	Factors associated with pneumonia in stroke	60
	Indian studies	60
	Justification of thesis	61
5	Methodology	62
6	.Data Analysis	69
7	.Results	70
8	.Discussion	99
9	.Limitations	104

10	Conclusions	105
11	Areas for future work	105
12	Bibliography	106
13	Annexures	114

## INTRODUCTION

Stroke is one of the most common and devastating disorder. It is one of the major non communicable diseases with significant morbidity and mortality along with cardiovascular disease and cancer. According to WHO Global health Observatory Data Repository, stroke is the second leading cause of death contributing to 12 percent of mortality worldwide.(1) It was responsible for 6.7 million deaths in 2012. It is a significant problem in lower income countries ranking fourth in the causes of death.

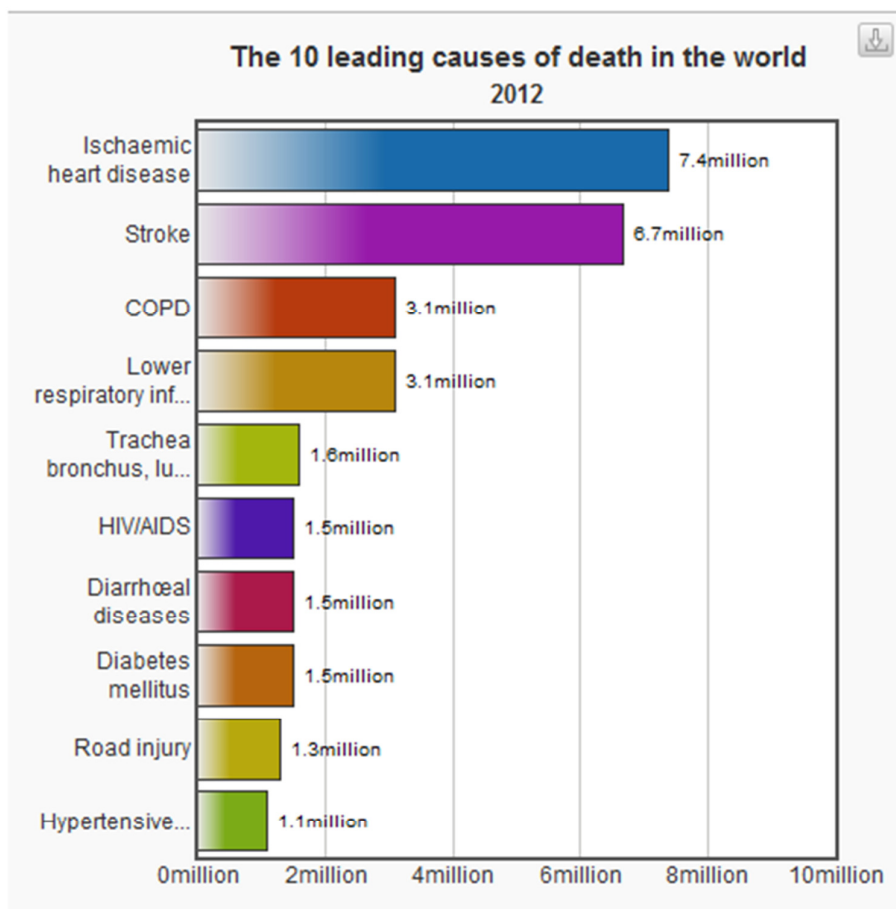


Figure 1: Leading causes of death in the world

In a review published in 2014 (2), data was compiled using all published systematic reviews on stroke and also WHO estimates of stroke related mortality for each country. In this study, it was found that the incidence varies across countries ranging from as low as 41 per lakh population in Nigeria to as high as 316 per lakh population in Tanzania.

The mortality estimates in stroke range from 16 to 23 percent in the first 30 days of stroke.(3) In this study conducted by Feigin et al, population based studies published between 1990 and 2003 were included and found that the overall mortality of stroke was 22.9%. This study also showed a decline in stroke incidence and mortality compared to 1970-80. WHO initiated MONICA project (Multinational Monitoring of Trends and Determinants in Cardiovascular Disease) in 1979 to study the trend of cardiovascular diseases over 23 years in 26 countries. As a part of this project, stroke registers were established in 10 countries. There was an overall decline in the incidence and stroke related mortality over years.(4)

Among several causes, pneumonia is the most common medical complication following stroke from second week onwards. One study looked at the causes of death in 1073 stroke patients and found that transtentorial herniation was the most common cause of death in first week. From the second week, pneumonia, sepsis and pulmonary embolism predominated. (5) Pneumonia is also the most common cause of fever within the first 2 days of stroke. In this study done by Grau et al, pneumonia was responsible for 40 percent of causes of fever in first 48 hours. (6)

Among several factors that contribute to pneumonia, dysphagia and aspiration are the most important.

This prospective cohort study is undertaken to find out the incidence of dysphagia and pneumonia in stroke patients admitted to medical wards in a tertiary care hospital and also to look at the factors associated with these complications.

## **AIMS OF THE STUDY**

1. To study swallowing dysfunction in stroke.
2. To study aspiration pneumonia in stroke, its relationship with dysphagia and outcomes.



## **OBJECTIVES OF THE STUDY**

1. To determine the incidence of swallowing dysfunction in stroke.
2. To study the factors associated with swallowing dysfunction in stroke.
3. To determine the incidence of aspiration pneumonia in stroke.
4. To determine factors associated with development of aspiration pneumonia in dysphagia.

## **REVIEW OF LITERATURE**

### **GLOBAL EPIDEMIOLOGY OF STROKE**

In 2013, stroke was the second most frequent cause of death after coronary artery disease, accounting for 6.4 million deaths (12% of the total) worldwide. (7) As a cause of death, stroke ranks fourth in low income countries, second in lower-middle and upper income countries and first in upper-middle income countries. Stroke is also a major cause of disability in the world. A study was done on Framingham cohort to see the disability in stroke survivors at six months.(8) This study found that 43 percent of elderly stroke survivors had moderate to severe neurological deficits. It was estimated that 47,943,000 DALYs were lost as a result of stroke worldwide. (9)

The incidence of stroke is rising in the developing countries and decreasing in the developed countries.(9) The Global Burden of diseases 2010 was published in 2014 which included 119 studies done between 1990 and 2010. (9) . Compared to 1990, the incidence, stroke related deaths and DALYs lost were high in 2013. In the year 2013, 16.9 million had first stroke, and there were 33 million stroke survivors. There were 5.9 million stroke related deaths and 102 million DALYs were lost. In 2010, the mean age of incidence was 74.5 years in high income countries and 69.4 years in low and middle income countries.

Low and middle income countries shared the maximum burden with 68.6 percent incident strokes, 52.2% prevalent strokes, 70.9% stroke deaths, and 77.7%

DALYs lost. The incidence of stroke increased by 12 percent in low to middle income countries and decreased by 12 percent in high income countries between 1990 and 2010. The ratio of mortality to incidence was 0.35. The geographical distribution of incidence is shown below.

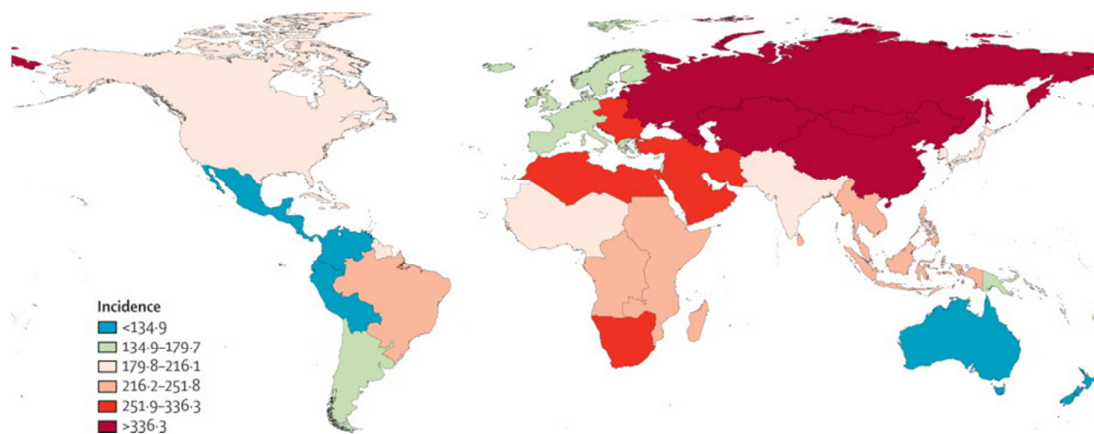


Figure 2: Geographical distribution of incidence of stroke

It is predicted that by 2030, there will be 12 million stroke deaths, 70 million stroke survivors and 200 million DALYs lost.

## EPIDEMIOLOGY OF STROKE IN INDIA

In India, the first study to detect annual incidence of hemiplegia was done in Vellore in 1970. This study showed an incidence of 13 per lakh population and prevalence of 42 per lakh. (10)

There are four population based stroke epidemiology studies conducted in India according to 'WHO-STEPS Stroke Protocol'- Kolkata, Bangalore, Trivandrum and Mumbai.

The Mumbai study (11) was published in 2008. This was conducted between Jan 2005 and Dec 2006 on 156861 people. A total of 456 strokes occurred giving an annual incidence rate of 145 per lakh. The age adjusted rate using Segi's 1996 world population was 152 per lakh. Males had an annual incidence rate of 149 per lakh and females had 141 per lakh. The mean age was 66 years. At the end of 28 days, there was 29.8 percent mortality.

The Kolkata study(12) was published in 2007. This study was conducted between March 2003 and February 2005 and included 52,377 people. This study found an age-standardized annual incidence rate of 145 per lakh. The prevalence was 545 per lakh population. The incidence and mortality was higher in women compared to men. The 30 day mortality rate was 41.08 percent. There was no difference between people living in slums and other areas.

The Bangalore study(13), published in 2009 found that stroke constituted 7 percent of admission to medical wards and 45 percent in neurological wards. The case fatality rate was 9 percent at the time of discharge and 28 day mortality was 20 percent. The mean age of presentation was 54.5 percent. In STEP 2, it included 23,312 people and found a proportional mortality of 6 percent.

The Trivandrum study(14) was conducted over 6 months in 2008 and studied 7,41,000 urban population and 1,85,000 rural population. A total of 541 strokes occurred giving an adjusted annual incidence rate of 135 per lakh population. The rate was same for rural and urban population. The 28 day mortality was 24.5 percent in urban population and 37.1 percent in rural population.

## DEFINITION OF STROKE

Traditionally, stroke was defined as “abrupt onset of neurological deficit that is secondary to focal vascular cause”. It is divided into two types- brain ischemia and brain hemorrhage. (7)

In 2014, the traditional definition has been updated to include both clinical and tissue diagnosis so that a uniform definition can be incorporated into research, clinical practice and measurement of public health. (9) In this AHA Expert consensus document, the following definitions have been proposed-

1. CNS infarction – “It is brain, spinal cord, or retinal cell death attributable to ischemia, based on pathological, imaging, or other objective evidence of cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution; or clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury based on symptoms persisting  $\geq 24$  hours or until death, and other etiologies excluded.”
2. Silent CNS infarction – “Imaging or neuropathological evidence of CNS infarction, without a history of acute neurological dysfunction attributable to the lesion.”
3. Stroke caused by intracerebral hemorrhage – “Rapidly developing clinical signs of neurological dysfunction attributable to a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma.”

Stroke is broadly classified into two kinds- ischemic and hemorrhagic.

There are classification systems proposed to classify stroke. One of them is TOAST classification which was developed for the Trial of Org 10172 in acute stroke treatment.(15) In this classification, strokes are classified into five kinds-

- Large artery atherosclerosis
- Cardioembolic stroke
- Small vessel occlusion
- Stroke of other determined aetiology
- Stroke of undetermined aetiology.

In NINDS classification (16), which was derived from Harvard stroke registry, the following groups are recognized-

- Infarction of unknown cause
- Infarction with normal angiogram
- Infarction in association with arterial pathology
- Cardioembolism
- Infarction due to atherosclerosis
- Lacunar infarct
- Intracerebral haemorrhage
- Other types

## ISCHEMIC STROKE

The ischemic stroke occurs secondary to blockage of blood flow to a part of brain so that the specific area is deprived of oxygen and nutrients. If the block lasts for more than a few minutes, there occurs irreversible damage to the brain. If the flow is restored immediately before this period, the symptoms will reverse and it is called transient ischemic attack. (17)

Once the infarction occurs, the region of brain shows two distinct areas- core and the penumbra. Penumbra is the area surrounding the infarcted cone, in which neurons have still not undergone necrosis. Once revascularization is done, this area can be saved. There are two pathways in which cell death can occur in infarction-

1. Ischemic pathway: In this pathway, due to the lack of oxygen and nutrients, mitochondria inside neurons fail to produce ATP. Due to lack of ATP, the cell membrane function is disrupted. This causes rise in intracellular calcium and activates proteolytic enzymes which break down membrane and cytoskeleton. The glutamate released from the nerve terminals activates the excitatory glutamate receptors which again activate postsynaptic pathways that cause cell break down. The generation of free radicals also contributes to cell death.

2. Apoptotic pathway: This pathway is important in the penumbra with lesser degrees of ischaemia. In this pathway, the mitochondria which are damaged release pro-apoptotic molecules like cytochrome c and apoptosis-inducing factor. These

factors activate enzymes called caspases which cause break down of cellular structures.

There are several factors which provide protection against necrosis. These are called neuroprotective factors. These include heat shock protein 70, Bcl-2 gene family, Prion protein, Neurotrophin-3, Interleukin-10, and Granulocyte colony stimulating factor.

Once the infarction occurs, it causes secondary effects over the surrounding brain due to edema. There are two mechanisms for edema- vasogenic edema and cytotoxic edema. The cytotoxic edema occurs immediately after stroke and is due to failure of cellular membrane pumps. This is reversible. The vasogenic edema occurs later and is due to increased permeability of capillary endothelial cells. This causes leakage of serum proteins and interstitial edema and increase in intracranial tension. This is responsible for displacement and cerebral herniation which can be fatal.(17)

The ischemic stroke can be due to thrombosis or embolism. Thrombosis of blood vessels occurs because of damage to endothelium of blood vessels by a variety of factors like hypertension, hyperglycemia, dyslipidemia and smoking. This damage leads to morphological abnormality called atherosclerosis which causes abnormal clotting of blood and blockage of the vessel in that area. When a thrombus circulates in the blood vessels, it is called an embolus. This embolus can originate from the major vessels like carotid artery or from the heart. (8)



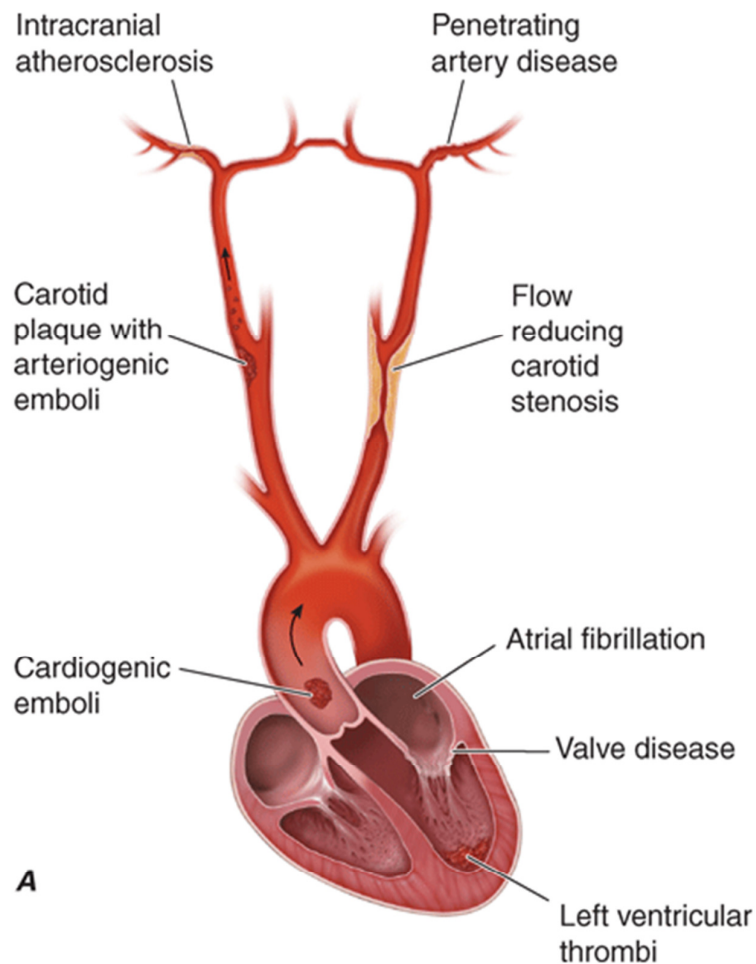


Figure 3: Pathophysiology of stroke

The common causes of cardioembolic strokes are atrial fibrillation, intracardiac mural thrombus post myocardial infarction, valvular heart diseases like mitral stenosis, presence of mechanical valves and infective endocarditis. Sometimes an embolus originating in the peripheral venous system enters the left heart through an atrial septal defect or patent foramen ovale. This is called paradoxical embolus. Atherosclerotic plaques in the major arteries like carotid bifurcation, aortic arch can also give rise to emboli.

There are several hypercoagulable states which increase the risk of thrombosis. Some of these are – Protein C and Protein S deficiency, Antithrombin 3 deficiency, Antiphospholipid antibody syndrome, Factor V Leiden mutation, malignancy, nephrotic syndrome, polycythemia vera and usage of oral contraceptive. Some of the other causes of strokes include vasculitis, fibromuscular dysplasia, Moyamoya disease and drugs like cocaine and amphetamine.(18)

Role of atherosclerosis- Atherosclerosis is a pathological process which causes obstruction of blood vessel lumens. This process includes deposition of lipid filled macrophages under the endothelium, which is called fatty streak. Later, there occurs accumulation of extracellular lipid which is covered by a layer of smooth muscle cells and collagen. This process is enhanced by hyperlipidemia, hypertension, hyperglycemia and smoking.

The initial trigger for atherosclerosis is injury to the arterial wall. The injury can be minimal causing alteration in the physiology of endothelial cells (called type I injury) or removal of endothelial layer with the deposition of platelets (called type II injury) or the damage can extend up to the deep intima causing thrombus formation (called type III injury). The injured endothelial cells express surface molecules for adhesion of lymphocytes and monocytes which migrate to the subendothelial surface and transform into foamy cells. The lipid that is deposited is engulfed by these monocytes and this lipid undergoes oxidation. The oxidation produces free radicals which cause further endothelial injury and changes in the

macrophages. The activated endothelial cells and macrophages release growth factors which promote smooth muscle cell proliferation and formation of plaque. The platelets get adhered at the site of endothelial injury and further release growth factors. Finally, the formation of thrombus that occludes the blood vessels occurs due to platelet and clotting factor activation at the site of atherosclerosis or bleeding into atheroma.(17)

## **HEMORRHAGIC STROKE**

The hemorrhage is divided into intracerebral hemorrhage and subarachnoid hemorrhage. Hemorrhage occurs due to disruption of blood vessel. This happens when the blood pressure increases putting stress over already weakened blood vessel due to atherosclerosis. (8)

The mechanism of neuronal damage in hemorrhagic stroke is due to the combined effect of ischaemia caused by disrupted vessel, irritation caused by the presence of blood and increased intracranial pressure.(17)

The causes of intracranial hemorrhage are- head injury, hypertensive bleed, cerebral amyloid angiopathy, liver disease, hemorrhagic transformation of infarct, bleed into tumour, Arteriovenous malformation, intracranial aneurysm, coagulopathy and dural AV fistula. (19)

## CLINICAL FEATURES OF STROKE

The signs and symptoms of stroke are varied. The clinical features depend on the part of the vascular system involved. Broadly, strokes can be classified into those occurring in the anterior circulation or posterior circulation. The Oxfordshire Community Stroke Project(20) classified strokes based on the part of circulation involved as-

- Total anterior circulation stroke
- Partial anterior circulation stroke
- Lacunar stroke
- Posterior circulation stroke

Occlusion of Anterior cerebral artery manifests as weakness of contralateral lower limb, abulia, transcortical motor aphasia, urinary incontinence and gait apraxia.(21)

Middle cerebral artery territory involvement varies according to the division of the artery involved. Involvement of superior division causes contralateral weakness, sensory loss, aphasias and apraxias. Involvement of inferior division causes superior quadrantonopsia and Wernicke's aphasia.

There is a difference between the clinical features of right and left cerebral hemisphere involvement. Involvement of right hemisphere causes disorders like neglect and inattention, apraxia and visuospatial disorientation. Large strokes

involving the right parietal cortex can cause anosognosia, in which the patient is unaware of the disability. Left hemisphere lesions cause abnormalities in language called aphasias. The various kinds of aphasias are - Wernicke's aphasia, Broca's aphasia, transcortical sensory aphasia, transcortical motor aphasia, conduction aphasia and global aphasia. Left hemisphere lesions also can cause a variety of apraxias.(21)

Posterior circulation strokes cause crossed hemiplegia with involvement of ipsilateral cranial nerves and contralateral hemiplegia. These are also associated with cerebellar symptoms, vertigo, dysarthria and rarely drop attacks. Several brain stem syndromes are described depending on the specific vascular territory involved. (21)

### **NIHSS SCORE**

NIHSS score is a quantitative measurement of neurological disability. It consists of 15 items which include testing awareness, cranial nerves, motor system, sensory system, cerebellar function, language and neglect. The minimum score is 0 while maximum is 42. The greater the score, the more is the severity of the stroke.

The score is also used to predict the outcome of stroke. Several studies are done which showed the usefulness of the score in this regard. In TOAST trial (22), published in 1999, 1281 patients were studied and a baseline NIHSS score was calculated. The patients were followed up after 7 days and 3 months and the primary outcomes were Barthel Index and Glassgow Coma Scale. In this study, it

was found that the outcomes worsen by 17 percent at 3 months with one point increase in NIHSS score. In patients with the score between 7 and 10, 46 percent had good outcome and in patients with score between 11 and 15, 23 percent had good outcome.

In another study (23), placebo group of National institute of neurological disorders and stroke rt-PA stroke trial was included and the outcome was compared to baseline NIHSS score. It was found that a score of more than 22 at 24 hours had a positive predictive value (PPV) of 98 percent to predict mortality. At 7 to 10 days, score more than 16 had a PPV of 92 percent.

In another study, NIHSS score was compared to Canadian neurological score, Middle cerebral artery neurological score and Guy's prognostic score and was found that NIHSS score provides the best prognostic information with a sensitivity of 71 percent and specificity of 90 percent.(24)

### **MODIFIED RANKIN SCALE**

This is used to assess disability. It consists of seven grades as follows-

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<b>Modified Rankin Scale (MRS)</b>	
0	No symptoms
1	No significant disability, despite symptoms; able to perform all usual duties and activities
2	Slight disability; unable to perform all previous activities but able to look after own affairs without assistance
3	Moderate disability; requires some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent, and requires constant nursing care and attention
6	Death

---

Figure 4: Modified Rankin Score

A study was done in 1995, in which 483 stroke patients were studied. Modified Rankin score was compared to various health aspects of daily living and it was found that the score is strongly associated with the disability in mobility, instrumental activities and living arrangements. The score was not correlating with cognitive and social functioning. So, modified Rankin score predicts disability in activities of daily living.(25)

A review was done to find out the appropriate scale to assess outcome in acute stroke trials. After assessing various scales for validity, relevance, cultural issues, language issues and training facilities, it was found out that modified Rankin score was the preferred outcome measure for trials involving acute stroke.(26)

## **THE PROBLEM OF DYSPHAGIA IN STROKE**

A meta-analysis which included 13 studies showed that the incidence of dysphagia ranges from 19 to 65 percent depending on the method of swallowing assessment used. (27) Mann et al(28) in 1999 evaluated the swallowing function of 128 stroke patients with clinical examination and Videofluoroscopic modified barium swallow. The incidence of dysphagia was 64 percent using Videofluoroscopic modified barium swallow and 51 percent using clinical examination.

In a study conducted by Smithard(29) in 2007, on 121 patients, 50 percent had dysphagia on clinical examination and 16.5 percent patients had aspiration. In 2012, Okubo et al(30) studied 50 stroke patients and found dysphagia in 32% of patients by clinical evaluation. He also studied relationship to NIHSS score and found that a cut-off score of 12 has 88% sensitivity and 85% specificity. In the same year, Baroni et al(31), studied 212 stroke patients and found 63% incidence of dysphagia by clinical evaluation.

In 2013, Flowers et al(32) reviewed charts of 221 stroke patients and found out 98(44%) had dysphagia by clinical examination. The median time to diagnosis was 2 days after stroke. The incidence of aspiration identified using Videofluoroscopic modified barium swallow studies, assessed during the acute stage of stroke, ranged from 30% to 51%.(27)



## NORMAL SWALLOWING

Normal swallowing is a complex process involving both voluntary and reflex activities. Oral cavity, pharynx and larynx are the structures involved in swallowing. Lips, cheeks and tongue are the main structures in oral cavity. The oral cavity is limited by faucial pillars. The pharynx is made up of constrictor muscles that originate on skull base, hyoid bone and thyroid cartilage anteriorly and insert into posterior median raphe. The lowermost muscle, cricopharyngeus arises from cricoid and acts as upper esophageal sphincter. The larynx lies anteriorly and has epiglottis, true and false vocal cords and has pyriform fossa on either side.(33)

The first description of swallowing was a three stage model comprising of oral, pharyngeal and esophageal stages.(34) Currently, two models are proposed- four stage model for liquids and the process model for solids.

**Oral preparatory phase (liquids):** The liquid bolus is held in the anterior part of the floor of the mouth and sealed posteriorly from oropharynx by approximation of tongue and soft palate.

**Oral propulsive phase (liquids):** The anterior part of the tongue rises up to touch hard palate and posterior part comes down and tongue squeezes liquid antero-posteriorly into pharynx.

**The process model (solids):** During swallowing of solid food, during mastication, part of food goes to oropharynx before the pharyngeal phase begins and bolus formation happens there. There are three stages-

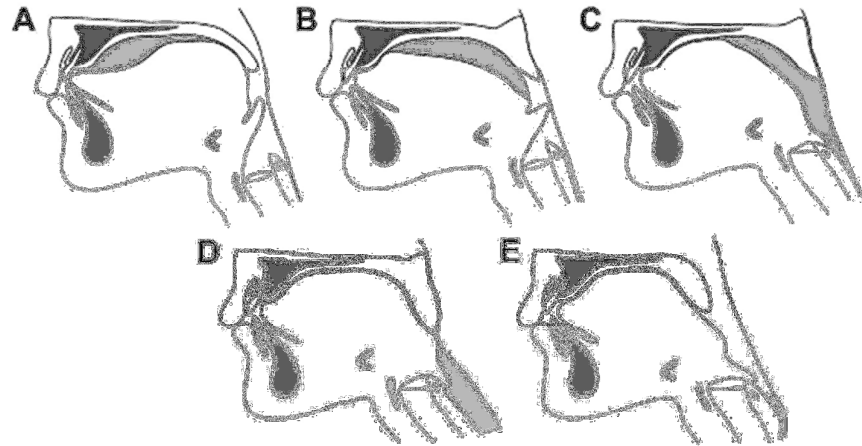
Stage1 transport: The food is taken to the post-canine region onto the occlusal surface of lower teeth for food processing by tongue.

Food processing: It includes chewing by cyclic movement of the jaw along with coordinated movements of the tongue, cheek, soft palate and hyoid bone. There is no sealing of oral cavity posteriorly.

Stage 2 transport: It is similar to oral propulsive phase. The food continues to accumulate and the bolus enlarges in the oropharynx.

**Pharyngeal phase:** It has two main components- propelling the food into esophagus through the pharynx and UES and protecting airway. The nasopharynx is sealed by soft palate. There are multiple mechanisms to prevent aspiration into larynx-the vocal folds seal the glottis, the arytenoids tilt forward to contact the base of epiglottis before opening of the UES, the hyoid bone and larynx are pulled upward and forward and larynx is tucked under the base of the tongue.

**Esophageal phase:** It consists of peristalsis assisted by gravity to transport food to stomach.(33)



**Fig 5. the diagram of swallowing a liquid bolus**  
**Normal swallowing of a liquid bolus:** Drawings based on a videofluorographic recording. (A) The bolus is held between the anterior surface of the tongue and hard palate, in a "swallow ready" position (end of oral preparatory stage). The tongue presses against the palate both in front of and behind the bolus to prevent spillage. (B) The bolus is propelled from the oral cavity to the pharynx through the fauces (Oral propulsive stage). The anterior tongue pushes the bolus against the hard palate just behind the upper incisors while posterior tongue drops away from the palate. (C-D) Pharyngeal stage. (C) The soft palate elevates, closing off the nasopharynx. The area of tongue-palate contact spreads posteriorly, squeezing the bolus into the pharynx. The larynx is displaced upward and forward as the epiglottis tilts backward. (D) The upper esophageal sphincter opens. The tongue base retracts to contact the pharyngeal wall, which contracts around the bolus, starting superiorly and then progressing downward toward the esophagus. (E) The soft palate descends and the larynx and pharynx reopen. The upper esophageal sphincter returns to its usual closed state after the bolus passes.

Figure 5: Swallowing of a liquid bolus

## DYSPHAGIA

Difficulty in swallowing is called dysphagia. It can be classified into oropharyngeal dysphagia and esophageal dysphagia. Oropharyngeal dysphagia is also called transfer dysphagia and it is difficulty in the initial part of swallowing. The causes of oropharyngeal dysphagia are classified according to the phase of swallowing affected.

Disorders effecting the oral preparatory phase are poor dentition, decrease in saliva production as occurs in Sjogren's syndrome, radiotherapy and use of anticholinergics, neurological dysfunction due to stroke, Amyotrophic lateral sclerosis, Parkinson's disease and local causes of mucosa like mucositis, aphthous ulcers or herpes infection.

The pharyngeal phase is effected by several neurological disorders like stroke, Amyotrophic lateral sclerosis, myasthenia gravis, muscular dystrophies and Parkinson's disease. Lack of coordination between pharynx and upper esophageal sphincter causes incomplete opening of the sphincter and dysphagia. Some of the local causes include cricopharyngeal bar, hypopharyngeal diverticulum and malignancies.(35) Stroke is one of the common causes of dysphagia and can be due weakness of tongue or laryngopharyngeal muscles or abnormalities in swallowing reflex. (36)

Dysphagia should be suspected in the following clinical settings (37)-

1. Altered sensorium – When a patient is drowsy, delirious or having dementia, swallowing dysfunction is likely to be present. The same is true for patients having inattention during eating or playing with food.
2. Dysphagia should be suspected when there are changes in the attitude towards food like avoiding eating in a group, avoiding fluids, eating for a long period or eating incompletely, changing head postures while trying to swallow or trying to take multiple small bites or several swallows.

3. The presence of bulbar dysfunction like dysarthria, gurgling or hoarse voice, drooling of food or saliva, oral residue after eating, presence of cough or choking while swallowing, clearing throat after swallowing or nasal regurgitation.

4. Sometimes, patients present with the complaints of sensation of food sticking in the throat, choking while eating, nasal regurgitation, profuse secretions, cough or dyspnea after eating or unexplained weight loss.

## SIGNIFICANCE OF DYSPHAGIA

Several studies have been done which showed that the presence of dysphagia was significantly associated with the development of pneumonia.(27) Presence of dysphagia was associated with an overall relative risk of 3.07 for development of pneumonia. All the studies used clinical evaluation for diagnosing dysphagia.

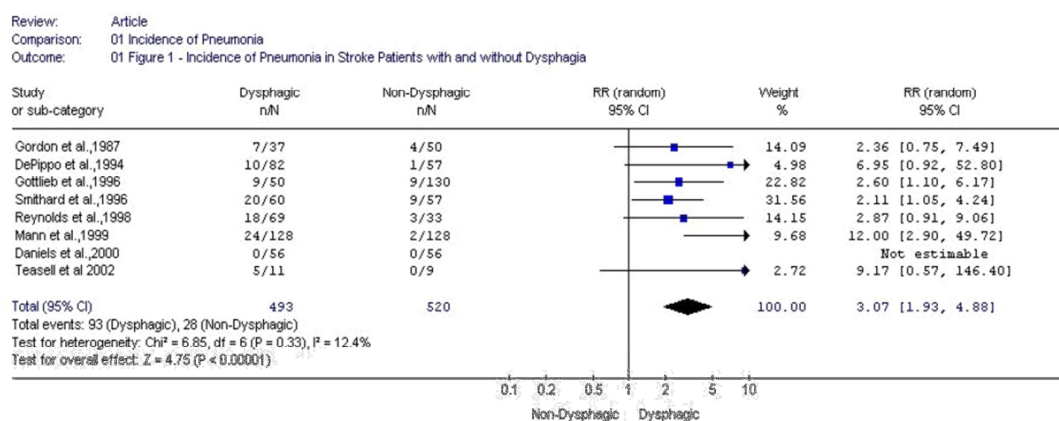


Figure 6: Relation between dysphagia and pneumonia

## **METHODS TO DIAGNOSE DYSPHAGIA AND ASPIRATION**

There are several methods to diagnose dysphagia and aspiration. These can be broadly divided into instrumental and non-instrumental.(27)

### Non instrumental Methods:

1. Clinical examination
2. Water swallowing test
3. Swallowing provocation test

### Instrumental methods

1. Videofluoroscopic Modified Barium Swallow
2. Endoscopic evaluation of swallowing
3. Pulse oximetry

### Clinical examination:

Bedside clinical examination is the most widely used practice. The clinicians look for drooling from mouth, muscle coordination, facial weakness, elevation of larynx, cough or throat clearing, change in voice after swallow.(38) Presence of gag reflex correlates poorly with safe swallowing and should not be used for decision making.(39) But, the clinical assessment has low sensitivity compared to

instrumental methods mainly due to failure to detect silent aspirators. Clinical assessment is also subjective and the interpretation varies according to the experience and expertise of the examiner. In a study of 469 patients, 276 were found to have silent aspiration. Elderly age, male gender and neurological illness were significantly associated with silent aspiration. (40)

The clinical methods can be patient symptom surveys, clinical assessments and functional assessments.

Among patient symptom surveys, Sydney swallow questionnaire is popular (Fig.7). This questionnaire consists of 17 questions which have to be answered on a visual analogue scale of 0-100. The questions cover the consistency of food associated with dysphagia, the anatomic region involved and type of dysfunction.(41) This score has been validated in patients with head and neck cancer(41) and oropharyngeal cancer(42).

There are other scoring system used especially in patients with head and neck cancer like - The Swallowing Questionnaire Quality of Life Questionnaire (SWAL-QOL and SWAL-CARE) and The MD Anderson Dysphagia Inventory (MDADI).

### SYDNEY SWALLOW QUESTIONNAIRE

1. How much difficulty do you have swallowing at **present**?
2. How much difficulty do you have swallowing **THIN** liquids do you have? (e.g. tea, juice, beer, coffee)
3. How much difficulty do you have swallowing **THICK** liquids? (e.g. milkshakes, soups, custard)
4. How much difficulty do you have swallowing **SOFT** foods? (e.g. ~~mornays~~, scrambled egg, mashed potato)
5. How much difficulty do you have swallowing **HARD** foods? (e.g. steak, raw fruit, raw vegetables)
6. How much difficulty do you have swallowing **DRY** foods (e.g. bread, biscuits, nuts)
7. Do you have any difficulties to swallowing your own saliva?
8. Do you ever have difficulty starting a swallow?
9. Do you ever have a feeling of food getting stuck in the throat when you swallow?
10. Do you ever cough or choke when swallowing solid foods? (e.g. bread, meat or fruit)
11. Do you ever cough or choke when swallowing liquids? (e.g. coffee, tea, water, beer)
12. How long does it take to eat an average meal?
13. When you swallow does food or liquid ever go up behind your nose or come out of your nose?
14. Do you ever need to swallow more than once for food go down?
15. Do you ever cough up or spit out food or liquids **DURING** a meal?
16. How do rate the severity of your swallowing problem today?
17. How much does your swallowing problem interfere with your enjoyment or quality of life?

Figure 7: Sydney swallowing questionnaire

Several screening tests were designed to identify patients with dysphagia following stroke who needed to be referred to speech therapists for detailed assessment. Some of the tests which were shown to have high sensitivity are – ‘Any two’ test, Gugging swallowing screen, Toronto Bedside swallowing screening test, Acute dysphagia screen and MetroHealth dysphagia screen.



In 'Any two' test(43), patients who are found to have two or more of the following features – abnormal phonation, dysarthria, poor voluntary cough, weak gag and change in voice after swallow, were considered to have dysphagia. This was shown to have a sensitivity of 92 percent compared to videofluoroscopic modified barium swallow.

The Gugging swallowing screen(44) is a scoring system consisting of preliminary part and direct swallowing test. In the direct swallow test, several consistencies are used like solid, semi-solid and liquids. The scores range from 0 to 20 and lower scores indicating dysphagia. The score was compared to Fiberoptic endoscopic evaluation of swallowing and the sensitivity was found to be 100 percent and a positive predictive value of 81 percent for aspiration.

In Toronto Bedside swallowing screen(45), four items were used – water swallowing test, sensations in pharynx, tongue strength and dysphonia. Presence of all the four had a sensitivity of 91 percent in predicting dysphagia on videofluoroscopic modified barium swallow.

Acute dysphagia screen(46) was developed in 2009. This consisted of five items – GCS, facial weakness, tongue weakness, palatal weakness and positive water swallowing test. This was validated against Mann's assessment of swallowing ability and was found to be 91 percent sensitive for predicting dysphagia and 95 percent sensitive for predicting aspiration.

MetroHealth dysphagia screen(47) was developed for use in emergency department by nurses. This consisted of five questions – whether patient is able to sit on his own upright for 10 minutes, the quality of voice, presence or absence of drooling, dysarthria and weak voluntary cough. This was compared to videofluoroscopic modified barium swallow and was found to be 95 percent sensitive.

Several studies used water swallowing test in which the patient is asked to swallow 100 ml of water at a time and the presence following findings were looked – choking, change in voice or desaturation on pulse oxymeter. The findings were used in various combinations and overall, the sensitivity ranged from 54 percent to 85 percent. Combination of all the three had a greater sensitivity. (27)

In swallowing provocation test, a catheter is inserted into nasopharynx through nose and water is injected in two stages – bolus of 0.4 ml followed by 2 ml. The water initiates an involuntary swallowing reflex. The outcomes looked at was – ‘latent period’, which is the time from giving bolus to initiation of swallow. This test was published from Japan and the latent period more than 3 seconds was found to have sensitivity of 75 percent to 100 percent to predict aspiration risk.(48)(49)

In 2012, Daniels et al (50) reviewed 16 studies published between 1985 and 2011 and looked at the components of screening tests which are most useful in predicting dysphagia and aspiration. Several factors were found to be associated with dysphagia and of those, the following were found to have sensitivity more than 80 percent – palatal weakness, abnormal water swallowing test, slurred

speech, weak voluntary cough, voice abnormality and impaired sensations in pharynx.

### **MANN'S ASSESSMENT OF SWALLOWING ABILITY**

This was developed by Dr. Giselle Mann by compiling the non-standardised clinical assessments used in Australia. This score is especially developed for stroke patients. An expert team of 15 was set up to assess factors which can be included and which need not and was psychometrically validated. Later, this was validated against previously well validated dysphagia screening tools and was found to have good content validity.(51)

This consists of 24 items with a total score of 200. They include Alertness, cooperation, auditory comprehension, control of respiration, respiratory rate, dysphasia, dyspraxia, dysarthria, drooling of saliva, lip seal, tongue movement, tongue strength, tongue coordination, oral preparation, gag reflex, palatal weakness, bolus clearance, oral transit, cough reflex, voluntary cough, voice, tracheal protection, pharyngeal phase and response.(51)

Later, the score was validated against videofluoroscopic modified barium swallow. A group of 128 stroke patients were included in the study and dysphagia was assessed by Mann's score and videofluoroscopic modified barium swallow. A score less than 180 was found to have 71 percent sensitivity in detecting dysphagia and 93 percent in detecting aspiration (27). This score has been recommended by University of Florida for assessment of swallowing.

In 2015, Jong-Chi h et al(52) compared Mann's assessment with videofluoroscopic dysphagia scale in 54 patients. A correlation coefficient of -0.509 was found between the two with high test-retest reliability and inter-observer reliability. This score has been used in several studies of dysphagia in stroke patients.(27)

## **FOIS SCORE**

This is the 'Functional Oral Intake scale'. It consists of seven scores as follows –

Level 1: Nothing by mouth.

Level 2: Tube dependent with minimal attempts of food or liquid.

Level 3: Tube dependent with consistent oral intake of food or liquid.

Level 4: Total oral diet of a single consistency.

Level 5: Total oral diet with multiple consistencies, but requiring special preparation or compensations.

Level 6: Total oral diet with multiple consistencies without special preparation, but with specific food limitations.

Level 7: Total oral diet with no restrictions.

This score was psychometrically evaluated and was found to have high inter-observer reliability (Cohen's kappa- 0.86-0.91) and high consensual validity. (53)

## **VIDEOFLUOROSCOPIC MODIFIED BARIUM SWALLOW**

Videofluoroscopy is considered the gold standard for swallowing assessments. In this technique, the patient is made to sit upright and asked to swallow radio-opaque barium. The barium is mixed with foods of various consistencies and given to the patient. It can be mixed with liquids, pudding, bread and biscuits. While the patient is swallowing, fluoroscopic images are obtained in lateral view. It can be recorded and played in slow motion to find out abnormal physiology and true aspiration. Exposure to radiation is a disadvantage in this technique.(38)

Various aspects of swallowing are noted as follows-

### **Oral Phase**

- Closure of lips
- Coordination and movement of tongue and bolus manipulation.
- Movements of soft palate
- Motion of the jaw
- Pocketing of food in oral cavity

### **Pharyngeal Phase**

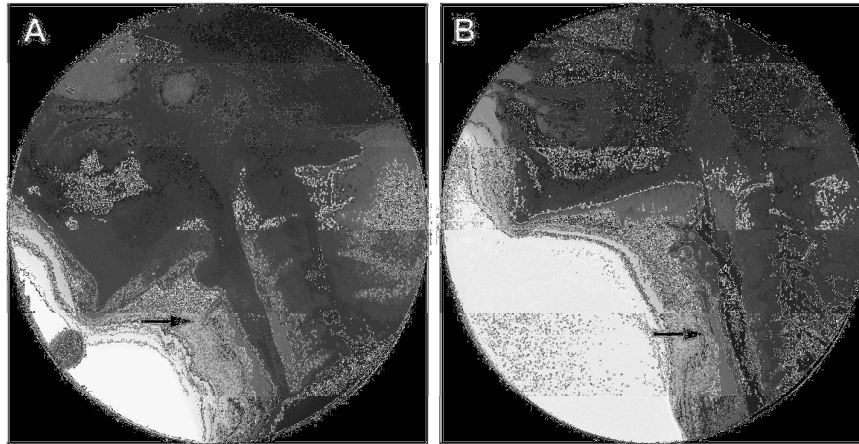
- Initiation, delay or absence of swallow
- Any residue in valleculae, pyriform sinuses. Regurgitation of food into nasopharynx.

## Laryngeal Function

- Elevation of larynx
- Penetration into laryngeal vestibule
- Aspiration
- Presence of cough and its effectiveness
- Vocal cord function

After Barium swallow, x ray is taken to see any chronic changes in the lungs or penetration of barium into tracheobronchial tree.(27)

The disadvantages of the procedure are – it is expensive and complex, needs training to interpret the results, involves radiation and it is difficult to perform in people who are not able to sit straight. There is also high inter-observer variability. But this test has been found to be cost effective when compared to bedside swallowing assessment or combination. This was thought secondary to detection of mild to moderate dysphagia and giving effective treatment which prevented pneumonia.(54)



**Fig 9. Penetration and Aspiration**

Videofluorographic images of laryngeal penetration (A) and aspiration (B) in dysphagic individuals swallowing liquid barium. Arrows indicate the leading edge of the barium in the airway.

Figure 8: Videofluoroscopy

## **FIBREOPTIC ENDOSCOPIC EVALUATION OF SWALLOWING**

Fibreoptic endoscopic evaluation of swallowing is another technique in which a small endoscope is passed through the nose and hypopharynx and larynx are directly viewed while patients swallow various foods. Topical anesthesia is given to decrease discomfort during the procedure. It also permits sensory testing. But, this technique evaluates mainly pharyngeal stage of swallowing with a brief period of 'white-out' during swallow.(55)

The advantages of this procedure are that it is less invasive, can be repeated, can be performed bedside and pharyngeal sensations can be tested. The disadvantages are - it needs skilled performer and needs costly equipment. Oral phase cannot be tested by this method.(38) The main complications associated with this procedure

are discomfort, epistaxis, laryngospasm, vasovagal reflex. The incidence of these complications is very low. (56) The procedure requires the combined effort of otolaryngologist, who performs the endoscopy and speech-pathologist, who does the swallowing assessment.

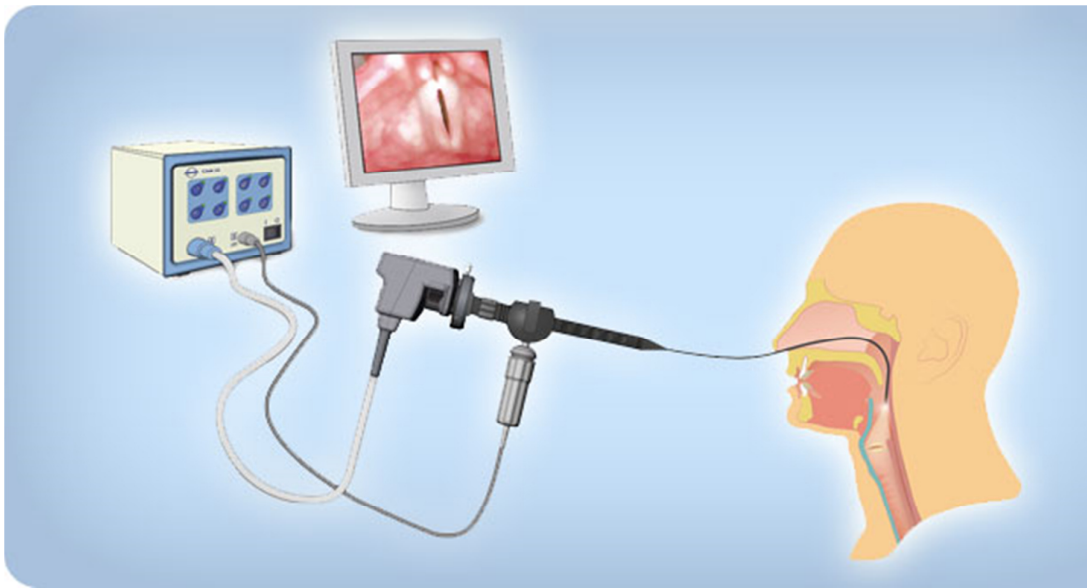


Figure 9: Fibreoptic Endoscopic Evaluation of Swallowing

The practice of using flexible endoscopes for assessment of dysphagia was started by Langmore et al in 1988. Later, several studies were done demonstrating the usefulness of endoscopy in the evaluation of dysphagia under multiple names like videoendoscopic evaluation of dysphagia and videoendoscopic evaluation of swallowing study.(57)



In 1998, Aviv et al (58) introduced sensory assessment during this procedure and called it FEESST- fiberoptic endoscopic evaluation of swallowing with sensory testing. The sensory deficits were classified into mild, moderate and severe deficits. When puree consistencies were used, 69% of patients with severe deficits had laryngeal penetration, where as 24% with normal or moderate deficits had laryngeal penetration ( $p < .001$ ).

In FEES, both static and dynamic examinations can be done. In addition, various compensatory postures may be tried and this can help to plan the rehabilitation strategy. The static examination is done with the tip of endoscope at three main positions- nasopharynx, upper position (at *velum palati*) and lower position (at laryngeal auditus). In upper position, vellaculae, pyriform fossae, interarytenoid area and laryngeal vestibule can be examined. In lower position, vocal cords can be tested.(55)

During dynamic testing, the patient is given a food bolus and asked to swallow. The main complication during this stage is inhalation of bolus which can be assessed initially by giving a small amount of water mixed with a dye like methylene blue and aspiration of the dye can be easily assessed. Inhalation of bolus can happen during any stage of swallow – pre-swallowing inhalation, intra-swallowing inhalation and post-swallowing inhalation. In pre-swallowing inhalation, when the patient is chewing the food, there can be premature drop of food into the pharynx and aspiration. The intra-swallowing inhalation cannot be directly seen due to the pharyngeal white-out. Post-swallowing inhalation occurs if

there are abundant food remains in the pharynx, velliculae which can be aspirated.  
(55)

There are several studies which compared FEES with modified barium swallow for assessment of aspiration. The first of these studies was published in 1988 by Langmore et al in which FEES was shown to have higher specificity for penetration and aspiration compared to the modified barium swallow.(57) In 1997, Wu et al (59) studied the two methods in 28 patients and found that there was an overall 14.5% disagreement between the two methods and FEES was more sensitive in detecting the risky features of swallowing such as aspiration, effective cough reflex and pharyngeal stasis compared to barium swallow. However, Leder and Karas (60) evaluated both procedures in seven pediatric patients and found 100% agreement between the two groups.

The difference in the outcomes between the population evaluated by FEESST and barium swallow was studied by Aviv.(58) He randomly assigned 126 patients to undergo either FEESST or barium swallow and followed them up over a period of 2 years. The primary end point was incidence of pneumonia and pneumonia free interval. He found that there was no statistically significant difference between the two groups.

## **ROLE OF SPEECH THERAPIST IN STROKE**

Speech therapists or speech-language pathologists are the health care professionals who are trained in the evaluation and management of communication and

swallowing disorders. (61) The practice of speech pathologists started in 1930s with their involvement in managing children with cerebral palsy. Later, around 1970-80, the field of speech pathology expanded forming a specialised branch. Currently speech pathologists are involved in various clinical settings as a part of multi-disciplinary team.(62)

In guidelines given by the American Speech\_Language\_Hearing Association, the role of speech pathologists is described as a part of diagnosing and treating cognitive-communication disorders and swallowing dysfunction after stroke. In patients with aphasia or dysphasia or dysarthria after stroke, speech pathologists use several strategies such as word retrieval, group sessions, structured discussions and role playing. The speech therapists also diagnose muscle weakness contributing to disordered speech and prescribe specific strategies to compensate for muscle weakness.

The speech pathologists also evaluate a patient's swallowing function and make recommendations including position changes, compensatory postures, prescribing diet consistencies and family education.(63) In another position statement released by American Speech-Language-Hearing association, speech language pathologists, who are trained, can perform endoscopy independently and the presence of physician is only required whenever there is a need to assess the anatomical abnormalities and functional evaluation of swallowing to give a medical diagnosis.(64)

## **PATHOPHYSIOLOGY OF DYSPHAGIA IN STROKE**

Using transcranial magnetic stimulation, it was found that swallowing is represented bilaterally but in an asymmetric manner. It was also found that stroke of dominant hemisphere more likely results in dysphagia and the severity depends on the amount of pharyngeal motor representation in the other unaffected hemisphere.(65)

Veis and Logemann(36) described Videofluoroscopic modified barium swallow findings in 38 stroke patients and found that swallowing abnormalities occur usually in combination rather than in isolation. Most patients exhibited delayed swallowing reflex followed by reduced pharyngeal peristalsis and reduced tongue control. Decreased laryngeal closure was seen only in brainstem strokes. Around one third of the patients aspirated, mostly because of delayed triggering of the swallowing reflex.

## **THE PROBLEM OF ASPIRATION PNEUMONIA IN STROKE**

Aspiration pneumonia is an important problem in stroke patients. In 2007, Sellers et al prospectively followed 412 patients over 3 months and found that pneumonia occurred in 18.9% of patients. (66) In a less rigorous study, based on population based registry, out of 13,279 stroke patients, 9% developed pneumonia during hospitalization. Pneumonia was associated with higher 30 day and one year

mortality. The association was mainly because of pneumonia, which was associated with higher mortality both after 30 days -adjusted MRR, 1.59 (95% CI, 1.31-1.93) and 1.76 (95% CI, 1.45-2.14) respectively. (67) The poor outcome of pneumonia was demonstrated in another study in which 8251 patients were prospectively studied and it was found that pneumonia occurred in 7.1% of patients and was associated with higher 30 day mortality (OR 2.2; 95% CI 1.8-2.7) and 1-year mortality (OR 3.0; 95% CI 2.5-3.7)(68)

### PATHOPHYSIOLOGY OF PNEUMONIA IN STROKE:

There are two theories explaining the pathophysiology of pneumonia in stroke- Aspiration theory and Stroke-induced immunodepression.(69)

#### Aspiration theory:

This is the traditional theory. Stroke patients have impaired swallowing function which causes aspiration while feeding. There are many studies linking the presence of aspiration to the development of pneumonia. (27)

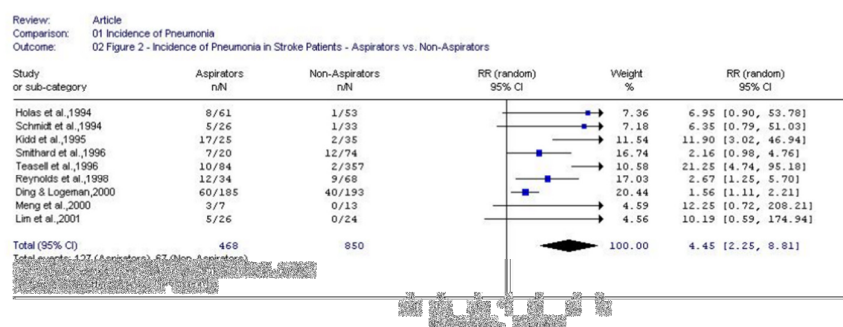


Figure 10: Relation between aspiration and pneumonia

Stroke patients also have aspiration of oral contents during sleep which was considered secondary to abnormality in dopamine transmission.(70) This was derived from an experiment in guinea pigs, in which D1 dopamine receptors were blocked which led to inhibition of swallowing reflex and decrease in the levels of substance P. Another study looked at the level of substance P in the sputum of elderly patients with aspiration pneumonia and found the levels to be low. Increasing the levels by using ACE inhibitors led to resolution of aspiration.(71)

But, the incidence of pneumonia in stroke was greater than that observed in other conditions which cause dysphagia or decreased consciousness. (72) Thus, it was considered that factors other than aspiration play a role in development of pneumonia in stroke patients.

#### Stroke-Induced immunodepression:

There is alteration of immunological function in stroke secondary to activation of 3 systems- sympathetic system, parasympathetic system and hypothalamo-pituitary-adrenal axis.(69)

Prass et al (73) conducted experiments in mice and found that stroke was associated with loss of lymphocytes, shift from Th1 to Th2 response, bacteremia and pneumonia. On blocking the beta receptors using propranolol, bacteremia was reduced and mortality was decreased. In another experiment, it was found that, in mice having cerebral ischemia, 200 CFUs of pneumococci were enough to cause

pneumonia whereas 2 lakh CFUs were required in control animals. This pneumonia was preventable by beta blockade.(74)

Parasympathetic system activation causes an increase in cholinergic activity which suppresses cytokine release. Paraventricular nucleus of hypothalamus was thought to be involved in the mechanism.

Stimulation of hypothalamo-pituitary-adrenal axis increases the circulating glucocorticoids which suppress lymphocytes.(69)

To summarise, pneumonia develops as a consequence of bacterial entry through aspiration and alterations in immunity which predisposes to infection.

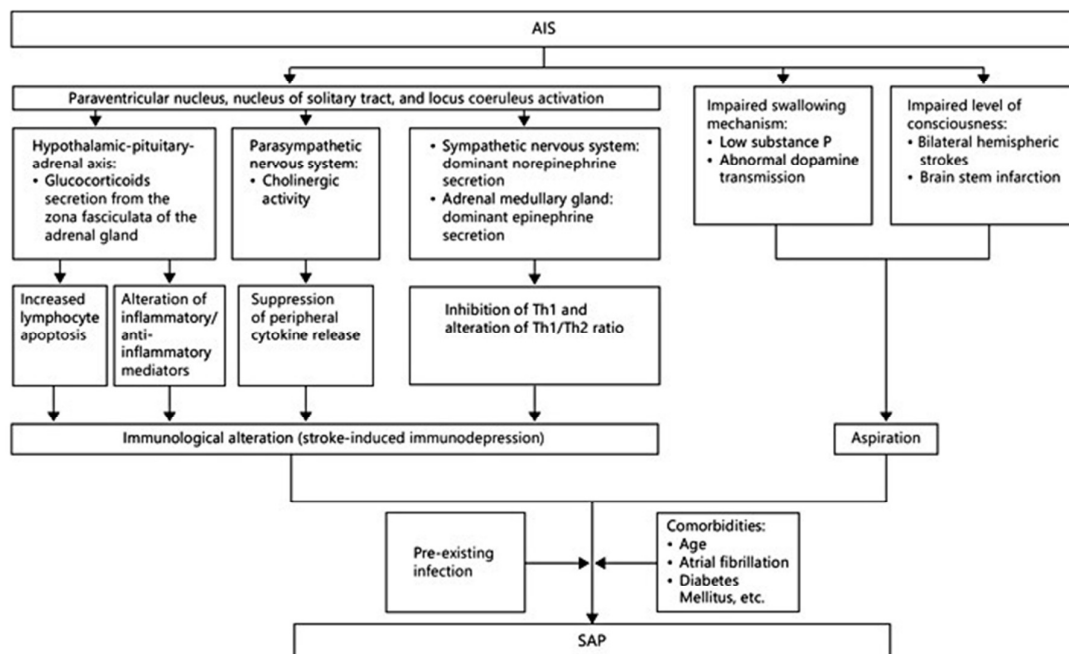


Figure 11: Pathophysiology of stroke associated pneumonia

## **FACTORS ASSOCIATED WITH PNEUMONIA IN STROKE**

Sellars et al (66) in 2007, studied 412 patients with stroke prospectively and found 18.9% of patients developed pneumonia. Ten parameters were assessed as predictors of pneumonia out of which five were significantly associated- elderly age (>65 years), dysphasia or dysarthria, modified Rankin score more than 4, abbreviated mental test score <8 and positive water swallowing test. Presence of 2 or more of these factors predicted pneumonia with 90.9% sensitivity and 75.6% specificity. Oral health was not an independent predictor of pneumonia.

In another study, 100 patients who were receiving Ryle's tube feeds due to dysphagia were followed over 100 days and was found that pneumonia occurred in 44% of patients. The independent predictors for pneumonia were low sensorium and severe facial palsy.(75)

## **INDIAN STUDIES**

The literature from India regarding dysphagia in stroke is sparse. One study conducted by Sundar et al (76) aimed to correlate vascular territory to the presence of dysphagia. They found that the incidence of dysphagia in patients with total anterior circulation infarct is 100%, 36% in Partial anterior circulation infarct, 18% in lacunar infarcts. One out of three posterior circulation infarcts had dysphagia. The study also looked at the factors predicting the development of pneumonia and found that the following factors have 100% positive predictive value for predicting chest infection- the consciousness level (GCS < 12), abnormal breathing pattern,



weak voice, absent/decreased tongue movements poor voluntary cough. Impaired gag reflex had sensitivity of 69% and specificity of 88% and low positive predictive value of 73% but good negative predictive value of 86%.

Another study by Radhakrishnan et al (77) looked at the usefulness of endoscopy to make a decision regarding resumption of oral intake. Sixteen patients were included in this study. All the patients were put on Ryle's tube as per protocol. After assessment by neurologists and speech therapists, decision was made to remove the Ryle's tube. These patients also underwent FEES and the final decision to remove the tube was made based on FEES findings. The initial decision to remove Ryle's tube was revoked in four patients after FEES.

## **JUSTIFICATION OF THESIS**

Considering the lack of adequate data from India on this problem and given the significant prevalence, morbidity and complications from the swallowing dysfunction in stroke, this study is undertaken.

A design of 'prospective cohort study' was chosen as the study is undertaken on a cohort of stroke patients and swallowing dysfunction, its prognosis and complications can be assessed during the follow-up.

## METHODOLOGY

### **RECRUITMENT:**

The study was conducted between August 2014 and May 2015. The patients were recruited from medical wards. The principal investigator screened the new admissions in the ward every day and stroke patients were identified. The patients were recruited as per the following inclusion and exclusion criteria.

- **Inclusion criteria:**

1. age-20 to 80
2. Stroke as per definition
3. Consenting for participation.

The definition of stroke is taken according to Harrison's principles of Internal Medicine 18e – 'A stroke, or cerebrovascular accident, is defined by abrupt onset of a neurologic deficit that is attributable to a focal vascular cause.'

- **Exclusion criteria:**

1. Prior swallowing difficulty
- 2 Refusal to consent
3. GCS <8/15.

**METHOD OF EVALUATION:**

Once the patients are recruited, the principal investigator documented the demographic and clinical details in the clinical research form. (Appendix) Age, address for communication, presence or absence of risk factors (diabetes, hypertension, smoking, alcohol) are noted through interviewing the patient or the relatives. The type and site of stroke are taken from the images in PACS. Then, clinical examination is done and the findings noted down. The score of the patients in various scoring systems like MUST, NIHSS, GCS, modified Rankin scale were calculated. FOIS is noted down with regard to the patient's ability to swallow water, banana or biscuit and at the same time Mann's assessment of swallowing ability was administered and the score of the patient is calculated. A cut-off of 178 was used to diagnose dysphagia.

**Assessment by speech therapist**

The request for speech therapist's assessment would then be kept in patient's chart with the study's account number written over it, which would be sent to the department of PMR through ward boys as a part of getting routine appointments. The study patients were identified based on the account number and those requests were sent to the speech therapists involved in the study. The speech therapists would come to the ward later and do a swallowing assessment and the findings are documented in a proforma. The speech therapist's assessment consists of initial

examination and followed by trial of oral feeds of various consistencies – water, honey, banana and pureed. The patients with swallowing difficulties were also taught exercises and compensatory postures. The patients are later visited by the speech therapists once a day and the reassessments were done with regard to improvement of swallowing, trial of consistencies and decision for starting oral feeds.

### **Fibreoptic Endoscopic Evaluation of Swallowing**

Some of the patients, who were able to sit, were referred for endoscopic evaluation of swallowing. This procedure was done in the department of ENT. During this procedure, a flexible nasal endoscope was inserted through the nose and the images were seen in a monitor. Trained otolaryngologists performed the procedure and a speech therapist was also present during the procedure. First, the oropharynx was inspected for any anatomical abnormalities and the following details were noted-

- Velopharyngeal closure
- Appearance of hypopharynx and larynx at rest
- Secretions and swallow frequency
- Base of tongue and pharyngeal muscles
- Vocal cords
- Sensory testing

After this, different consistencies of food were given to the patients adding blue food coloring agent, to look for the presence of aspiration into larynx. The substances used were-

- Solid – banana
- Thick liquid – honey
- Thin liquid – cerelac powder mixed with water by the speech therapist

It was also noted whether the patients had any nasal bleeding after the procedure.

#### **FOLLOW UP:**

The patients were followed up during hospital stay every day and clinical examination was performed and the following investigations were done at the sign of fever, cough —total count, differential count and chest x-ray. The treatment decisions were made by the treating team.

The following definition was used for diagnosing aspiration pneumonia during hospitalisation (Carnaby et al 2006)-

At least three of :

- a) fever more than 38°C;
- b) productive cough;

- c) abnormal respiratory examination (tachypnoea >22 breaths per min, tachycardia, inspiratory crackles, bronchial breathing);
- d) arterial hypoxaemia ( $\text{PaO}_2 < 9.3 \text{ kPa}$ );
- e) culture of a relevant pathogen; and
- f) positive chest radiograph in a patient with suspected chest infection.

At the time of discharge, the patients were advised to follow up in stroke clinic.

After discharge, the patients were periodically followed up by telephone and asked regarding the intake of food, removal of Ryle's tube- whether removed by self or in the hospital, any symptoms of aspiration like cough while swallowing and clearing throat and the swallowing ability was scored by FOIS score. The patients were also enquired regarding symptoms of fever, cough and any hospital admissions or usage of antibiotics. If the patients died, it was enquired whether the patient had any fever, cough and breathing difficulty at the time of death. A death was considered secondary to pneumonia if the patient had any of the above symptoms.

#### **DATA SOURCES/MEASUREMENT:**

The following data is taken from the patient directly- demographic details, clinical examination findings, NIHSS score, FOIS score, modified Rankin score, GCS score, MUST score, Mann's score.

The following data is taken from PACS- type of stroke, location of the stroke, chest x-ray findings.

The findings during clinical evaluation of swallowing and endoscopic assessment are recorded in a pre-specified proforma and data is taken from it.

The follow up is for duration of 3 months. The following information is collected during follow up-

- Development of pneumonia
- Day of development of pneumonia post stroke
- Day of death post stroke
- Removal of Ryle's tube- self/hospital
- Day of Ryle's tube removal
- FOIS score at discharge at 1 month, 2 months and 3 months.

The data was initially entered in the clinical research form. The data entry into the computer was done using Epidata and the analysis was done using SPSS.

#### **SAMPLE SIZE:**

The sample size is calculated using the formula- 
$$\frac{[Z(1-\alpha)]^2 \times P \times Q}{D^2}$$

### Single Proportion - Absolute Precision

Expected Proportion	0.13	0.13
Precision (%)	2	5
Desired confidence level (1- alpha) %	95	95
Required sample size	1086	174
10% loss to follow up	1207	193
20% loss to follow up	1358	218

The required sample size to show the incidence of pneumonia of about 13% with precision of 5% was found to be 200 subjects with 95% confidence limits with 10% loss to follow-up.

### DATA ANALYSIS:

The data was analyzed for demographic details, the presence of dysphagia, the improvement in swallowing function, death or development of pneumonia, duration of Ryle's tube feeding.

There are four sets of data:

1. Details regarding stroke and scoring systems.
2. Clinical evaluation of swallowing function.
3. Endoscopic evaluation
4. Follow up data.

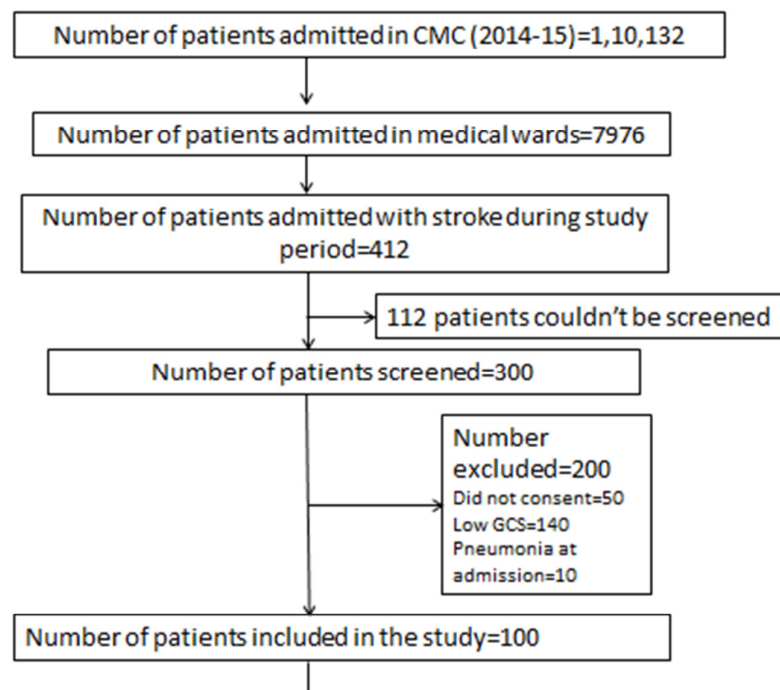


**STATISTICAL ANALYSIS:**

The prevalence was calculated as a percentage and the 95% confidence interval was also calculated. The descriptive measures were expressed as frequencies and percentages for categorical variables and using mean and standard deviations for continuous variables. The associated risk factors were assessed using a Fisher's exact test and then followed by a Logistic regression analysis for those variables that are significant using Fisher's exact test.

## RESULTS

During the study period from August 2014 to April 2015, a total of 1,10,132 patients were admitted in this hospital under all the departments. Under the department of Medicine, 7976 patients were admitted. Among these patients, 412 patients were admitted with stroke. 300 patients could be screened for inclusion into the study. Of these, 140 patients were excluded because of low GCS. 10 patients had pneumonia at presentation and 50 patients did not consent to participate in the study. So, 100 patients were included in the study. (Figure 12)



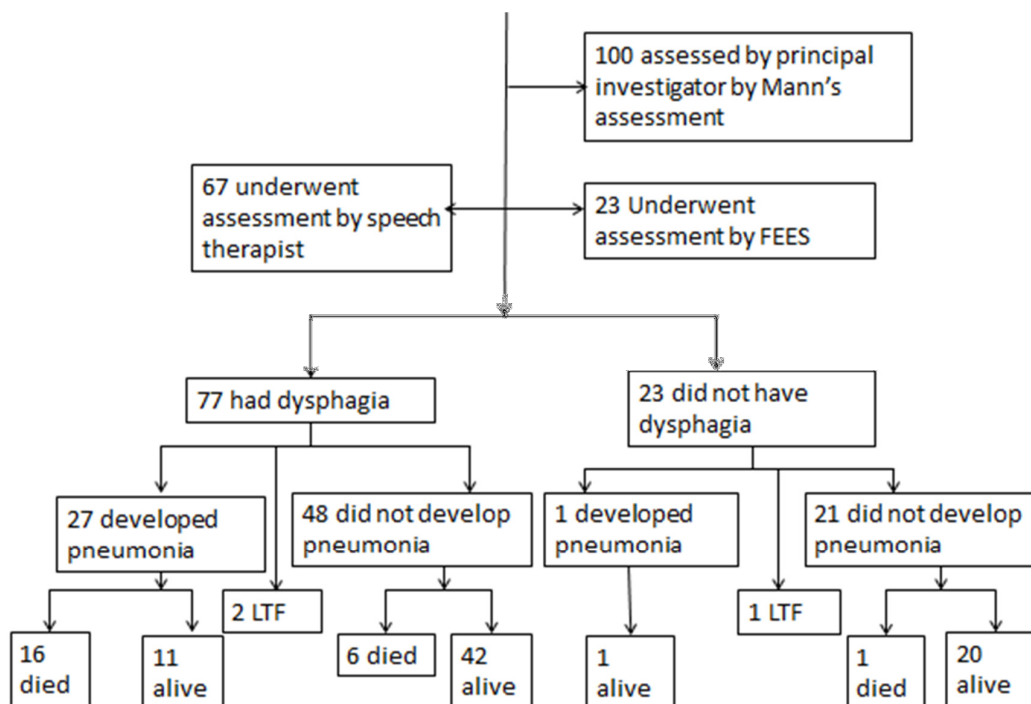


Figure 12: STROBE statement

\* LTF= Lost to follow up.

## 1. Admission characteristics of the patients:

### 1a. Demographic details:

The majority of patients included in the study were males, with a mean age of around 56 years. (Table 1)

N=100		
Age (Mean)	56.33 ;SD14.288	Median=55
Sex		
Male	66	66%
Female	34	34%

Table 1: Demographic details

### 1b. Risk factors:

Majority of patients had diabetes mellitus and hypertension. Smoking and alcoholism were observed in a few patients. (Figure 13)

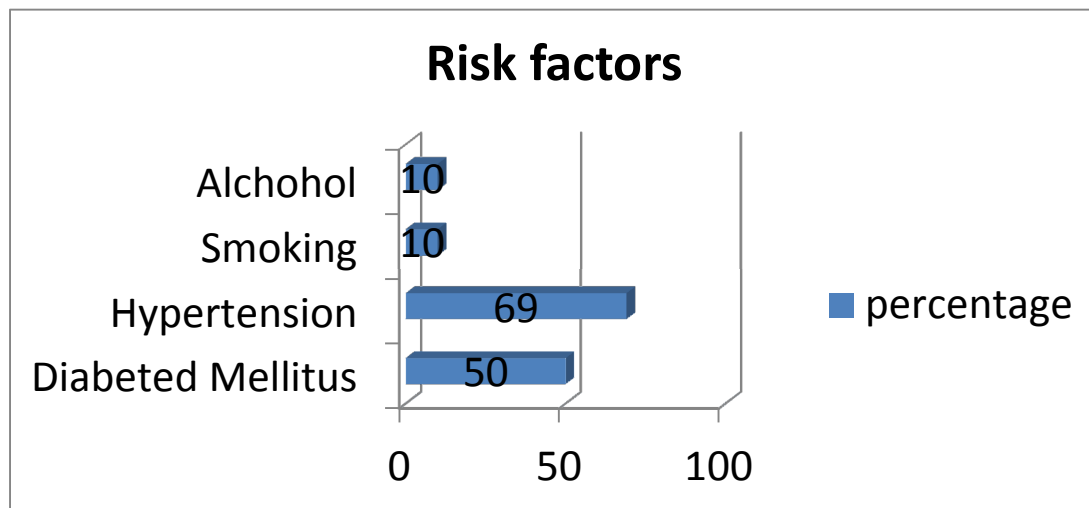


Figure 13: Risk factor profile of patients

### 1c. Clinical features:

Most of the strokes were ischemic (72%). Of the patients having ischemic strokes, 60 percent had Partial anterior circulation infarct followed by posterior circulation infarcts (9 percent) and lacunar infarcts (3 percent). None of the patients had Total anterior circulation infarcts. In patients having haemorrhagic strokes (28%), the most common site was basal ganglia followed by thalamus. (Figure 14)

Left sided strokes (57%) were slightly more in number compared to the right side strokes (41%). 2 patients had bilateral involvement in brain stem. Majority of the patients had facial weakness (79 %) and weak gag reflex (66 %) at admission.

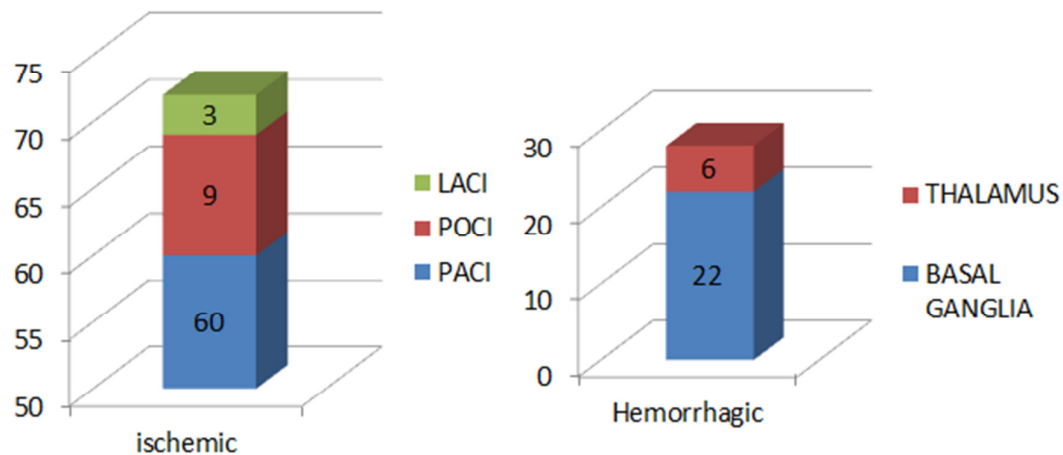


Figure 14: Location of stroke

Most of the patients had moderate severity of stroke with a median NIHSS score of 11 and a predicted 30 day mortality of 13.9 percent. (78) Most of the patients were in good consciousness with a median GCS score of 15. Majority of the patients were at low risk of malnutrition, as assessed by MUST score (median-0). Most of the patients had moderate disability with a modified Rankin score of 4. The baseline swallowing function was poor in majority of patients with a median FOIS score of 2. The median Mann's score was 163. (Table 2)

	Median (IQR)
NIHSS score (0-42)	11 (6-15.75)
GCS	15 (11.25-15)
E	4 (4-4)
M	6(6-6)
V	5(4-5)
mRS (0-6)	4 (3-4)
Mann's score(0-200)	163 (130-186.5)
FOIS score (1-7)	2 (1-5)
Facial nerve	79 had weakness

Table 2: Baseline characteristics

## 2. Dysphagia:

### 2a: Clinician's assessment of dysphagia

Out of 100 patients, 68 had dysphagia as assessed by clinical evaluation using Mann's assessment of swallowing ability. (Figure 15) A cut-off of 178 was used to diagnose dysphagia. Severity of dysphagia was graded according to the Mann's score. 47.06 percent of patients had severe dysphagia (Mann's score <138). Moderate dysphagia (Mann's score 167-137) was seen in 30.88 percent and 22.08 percent had mild dysphagia (Mann's score 167-178). (Figure 16)

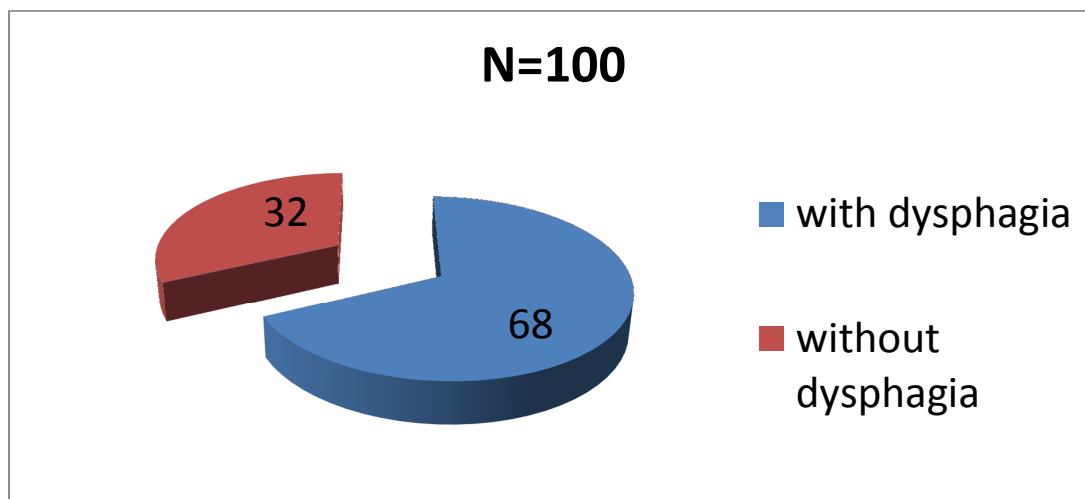


Figure 15: Incidence of dysphagia as assessed by Mann's assessment of swallowing ability

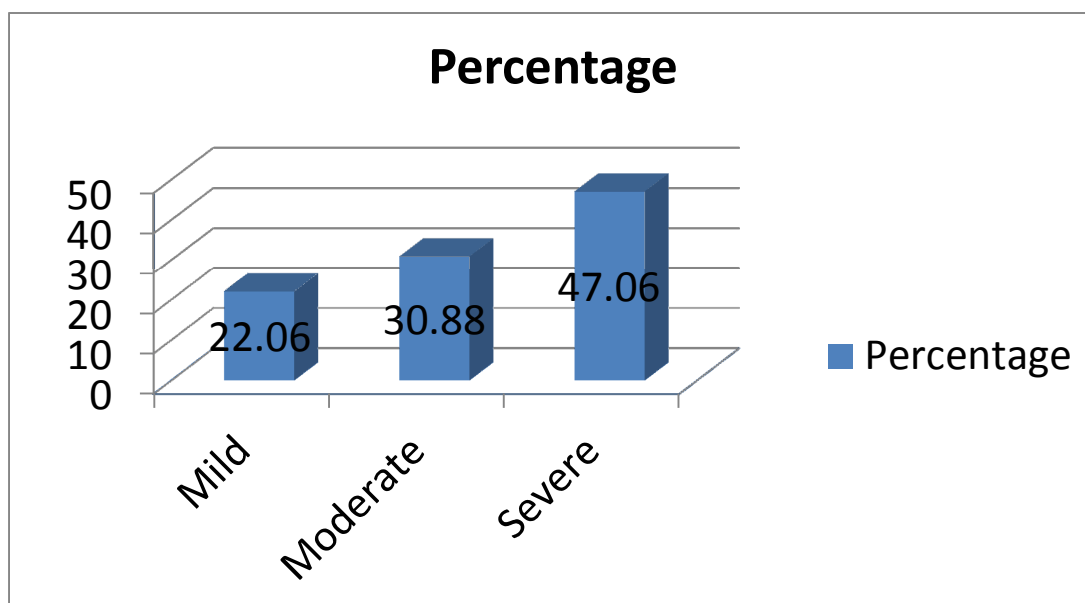


Figure 16: Severity of dysphagia as assessed by Mann's assessment of swallowing ability

## 2b. Speech therapist's assessment of dysphagia

65 patients underwent assessment by speech therapist. The clinical findings observed during assessment are tabulated (Table 3). The major findings observed were abnormality of movement of lips, tongue and jaw, abnormal voice, diminished gag reflex, poor cough and abnormal dry swallow.

	<b>Abnormal</b>	<b>Percentage</b>
Position of lips at rest	20	30.7
Lips movement	53	81.5
Position of tongue at rest	12	18.4
Tongue movement	51	78.4
Position of jaw at rest	8	12.3
Jaw movement	49	75.3
Gag reflex	35	53.8
Pharyngeal sensations	8	12.3
Voice	58	89.2
Quality of cough	28	43
Dry swallow	31	47.6

Table 3: Clinical findings during speech therapist's assessment of swallowing

Out of 65 patients, speech therapist diagnosed dysphagia in 50 patients. (Figure 17)



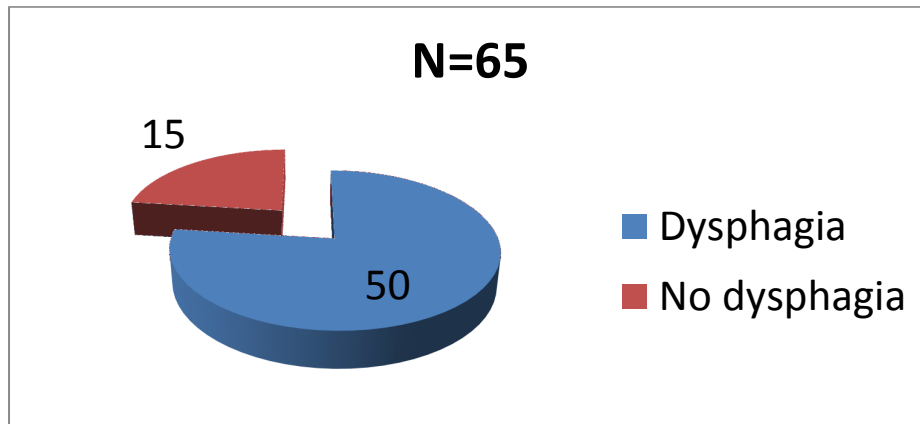


Figure 17: Speech therapist's diagnosis of dysphagia

Comparing the diagnosis of dysphagia made using Mann's score and speech therapist, the clinician's assessment had 100% specificity, but the sensitivity was 80.2 %.(Table 4)

Clinician/speech therapist	Dysphagia present	Dysphagia absent
Dysphagia present	41	0
Dysphagia absent	9	15

Table 4: Comparison between clinician's and speech therapists assessment of dysphagia

### 2c. Assessment using Fiberoptic endoscopic assessment of swallowing

21 patients underwent Fiberoptic Endoscopic Evaluation of Swallowing. The major findings observed were pooling of secretions in hypopharynx, vocal cord palsy and aspiration.(Table 5) Five patients had normal findings during FEES, but

were found to have dysphagia clinically with Mann's scores of 163, 156, 185, 188 and 160.

	Normal	Abnormal (Percentage)
Velopharyngeal closure	18	3 (14.2)
Pooling of secretions in hypopharynx	13	8 (38)
Base of tongue	21	0
Medialisation of pharyngeal wall	17	4 (19)
Vocal cords	12	8 (38)
right side		5
Bilateral		3
Aspiration	12	8 (38)

Table 5: Assessment of swallowing using Fibreoptic Endoscopic Evaluation of swallowing

## 2d. Final diagnosis of dysphagia

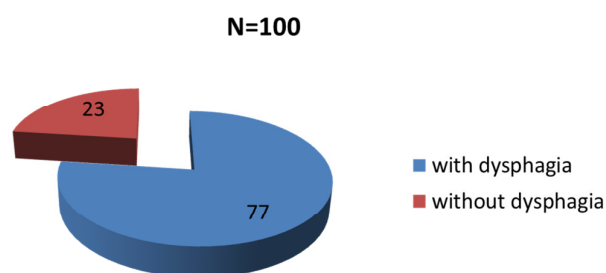


Figure 18: Number of patients with dysphagia using combined assessment with Mann's score, speech therapist's assessment and FEES

Finally, a diagnosis of dysphagia was made taking into consideration all the three assessment tools. A total of 77 patients were diagnosed to have dysphagia.(Figure 18)

An ROC curve constructed to assess the performance of Mann's score showed an area under curve (AUC) of 0.99. A cut-off point of 180 showed 100 percent % and 88% specificity. Best sensitivity and specificity of 96% and 90% were found with a cut-off of 181.(Figure 19)

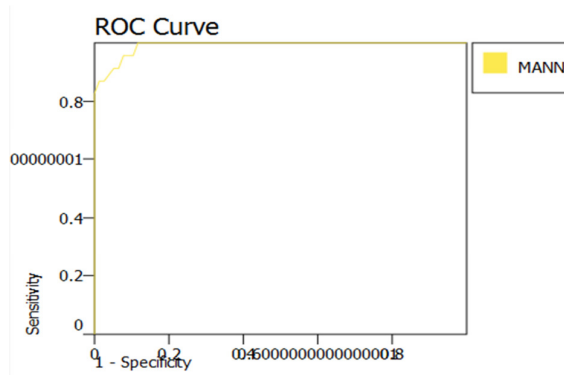


Figure 19: ROC curve assessing performance of Mann's score in diagnosing dysphagia

### 3. Factors associated with dysphagia:

In this study, dysphagia was present in females more than males which was statistically significant. Presence of risk factors for stroke like diabetes mellitus, hypertension, and alcoholism did not have significant influence on the development of dysphagia. However, there is a statistically significant difference

between the number of smokers in the two groups and the group without dysphagia had more number of smokers.(Table 6)

Variable	With dysphagia(n=77)	Without dysphagia(n=23)	P value
Sex(%)			<b>0.02</b>
Males(n=66)	46(69.7%)	20(30.3%)	
Females(n=34)	31(91.1%)	3(8.82%)	
Diabetes Mellitus(%)	38 (49.35%)	12(24%)	0.818
Hypertension(%)	52(67.53%)	17(73.9%)	0.618
Smoking(%)	5 (6.49%)	5(21.74%)	<b>0.047</b>
Alcohol(%)	7(9.09%)	3(13.04)	0.692

Table 6: Effect of sex and the risk factors of stroke on development of dysphagia

There was no significant difference between ischemic and haemorrhagic strokes in the development of dysphagia. The site of stroke and the side of hemisphere involved did not have significant influence on the development of dysphagia. Presence of facial palsy was significantly associated with the presence of dysphagia. (Table 7)

Variable	With dysphagia(n=77)	Without dysphagia(n=23)	LR	df	P value
Ischemic stroke	53(68.8%)	18(78.2%)			0.443
Hemorrhagic stroke	24(31.1%)	5(21.7%)			
Site of stroke			5.62	4	0.331
PACI	46(59.7%)	14(60.8%)			
LACI	2(2.6%)	1(4.3%)			
POCI	5(6.4%)	4(17.3%)			
basal ganglia	18(23.3%)	4(17.3%)			
thalamus	6(7.7%)	0			
Side of stroke					0.630
Right	30(40%)	11(47.8%)			
left	45(60%)	12(52.1%)			
Abnormal facial nerve	67(87%)	12(52.1%)			<b>0.001</b>

Table 7: Association of clinical features of stroke with dysphagia

The mean age was similar in both groups of patients. Both groups of patients were at low risk of malnutrition as assessed by MUST score. The patients having dysphagia had more severe stroke compared to those without dysphagia as assessed by NIHSS score (12 vs 4) (see Appendix). Though the median GCS was similar between the two groups, the mean value was significantly different with eye and verbal components of GCS being significantly low in the group having dysphagia. Most of the patients with dysphagia were dependant for mobility and ADLs with a median modified Rankin score of 4 while those without dysphagia were independent with a score of 2. The median Mann's score was 153 in those with dysphagia and 198 in those without dysphagia. The median FOIS score (see Appendix), which assesses the functional ability, was significantly different between the two groups with the patients having dysphagia had lower score of 2 compared to a score of 7 in those without dysphagia. (Table 8)

Variable	With dysphagia(n=77)	Without dysphagia(n=23)	P value
Age (Median (IQR))	58(47-67)	51(46-60)	0.182
MUST score (Median (IQR))	0 (0-1)	0	0.117
NIHSS score (Median (IQR))	12(9-16)	4(3-8)	<b>0.000</b>
GCS (Median (IQR))	15(10-15)	15	<b>0.001</b>
Modified Rankin score(Median (IQR))	4	2(1-3)	<b>0.000</b>
Mann's score(Median (IQR))	153(126-170)	198(191-200)	<b>0.000</b>
FOIS score (Median (IQR))	2(1-3)	7(6-7)	<b>0.000</b>

Table 8: Association of various scoring systems with dysphagia

In the swallowing assessment done, the following findings on clinical examination were associated with the presence of dysphagia - weakness of lips, weak movement of jaw and weakness of tongue. Presence of weak gag reflex, and poor cough strength were significantly associated with dysphagia. Inability or slowness in dry swallow (swallowing one's own saliva) was associated with dysphagia. The symmetry of lips and tongue at rest, decreased pharyngeal sensations and the quality of voice were not associated with dysphagia. (Table 9)

Variable	With dysphagia(n=50)	Without dysphagia(n=15)	P value
Abnormal symmetry of lips at rest	18(36%)	2(13.3%)	0.12
Abnormal movement of lips	48(96%)	5(33.3%)	<b>0.000</b>
Abnormal symmetry of tongue	11(22%)	1(6.6%)	0.26
Abnormal movement of jaw	46(92%)	3(20%)	<b>0.000</b>
Abnormal Strength of tongue	46(92%)	5(33.3%)	<b>0.000</b>
Weak gag reflex	33(66%)	2(13.3%)	<b>0.001</b>
Sensory examination	8(16.3%)	0	0.18
Abnormal cough strength	28(56%)	0	<b>0.000</b>
Abnormal dry swallow	31(62%)	0	<b>0.000</b>

Table 9: Clinical swallowing assessment for dysphagia

When the swallowing ability was tested using thin liquids, the presence of delayed initiation of swallow and presence of oral residue were associated significantly for the presence of dysphagia. The presence of weak lip seal, drooling, delayed elevation of larynx, cough after swallow, clearing the throat after swallow or wet voice or any change in voice after swallow were not significantly associated with pneumonia.(Table 10)

	<b>With dysphagia(n=13)</b>	<b>Without dysphagia(n=19)</b>	<b>P value</b>
Abnormal lip closure	2(10.5%)	0	0.50
Drooling	2(10.5%)	0	0.50
Abnormal mastication	3(15.7%)	0	0.25
Abnormal initiation of swallow	8(42.1%)	0	<b>0.01</b>
Laryngeal elevation abnormal	4(21%)	0	0.12
Cough after swallow	4(21%)	0	0.12
Change in voice quality present	1(5.26%)	0	1.0
Wet phonation	1(5.26%)	0	1.0
Voluntary throat clearance	1(5.26%)	0	1.0
Oral residue present	8(42.1%)	0	<b>0.01</b>

Table 10: Swallowing assessment using thin liquids

When thick liquid was used for assessment, none of the examination features showed significant difference between the two groups.(Table 11)

	<b>With dysphagia (n=28)</b>	<b>Without dysphagia(n=1)</b>	<b>P value</b>
Abnormal lip closure	1(3.5%)	0	1
Drooling	2(7.14)	0	1
Abnormal mastication	2(7.14%)	0	1
Abnormal initiation of swallow	15(53.5%)	0	0.80
Laryngeal elevation abnormal	11(39.29%)	0	1
Cough after swallow	5(17.8%)	0	1
Change in voice quality present	-	-	
Wet phonation	-		
Voluntary throat clearance	2(7.14%)	0	1
Oral residue present	17(60.7%)	0	0.93

Table 11: Swallowing assessment using thick liquids



Fibreoptic Endoscopic Evaluation of Swallowing was done in 24 patients. 3 patients were not cooperative for examination. Of the rest, 15 patients had dysphagia and 6 patients did not have dysphagia. The features significantly associated with dysphagia were the pooling of secretions in the hypopharynx and aspiration. (Table 12)

	<b>With dysphagia(n=15)</b>	<b>Without dysphagia(n=6)</b>	<b>P value</b>
Abnormal velopharyngeal closure	3(20%)	0	0.52
Pooling of secretions in hypopharynx	8(53.3%)	0	<b>0.04</b>
Abnormal base of tongue	0	0	
Medialisation of pharyngeal wall present	4(26.6%)	0	0.28
Vocal cord palsy			Chi 2 2.3 df-2, 0.31
Unilateral	4(28.5%)	1(16.6%)	
Bilateral	3(21.4%)	0	
Aspiration	8(57.1%)	0	<b>0.04</b>

Table 12: Fibreoptic Endoscopic Evaluation of Swallowing for dysphagia

### **3a. Independent factors predicting dysphagia**

Using logistic regression, weak gag reflex was the only independent factor predicting pneumonia. (Table 13)

	OR	SE	P value
Facial Nerve	0.67	1.15	0.56
NIHSS	0.42	0.57	0.09
mRS	0.38	0.57	0.50
Gag reflex	3.04	1.12	0.007

	OR	SE	P value
Lip strength	0.01	29000	1.0
Tongue strength	0	29291	1.0
Gag	0.17	1.29	0.16
Strength of cough	0	5874	0.99
Dry swallow	0	4214	0.99

Table 13: Logistic regression for factors predicting dysphagia

#### 4. Prognosis of dysphagia:

Dysphagia improved in a majority of patients over three months. In patients with mild dysphagia, a mean FOIS score of 7 was reached by 1 month, whereas in patients with moderate dysphagia, the score was reached by 3 months. In patients with severe dysphagia, the mean FOIS score was 6 at the end of 3 months. 9 patients were dependent on Ryle's tube at the end of 3 months and 9 patients had limitation to one or more consistencies at the end of 3 months. (Figure 20)

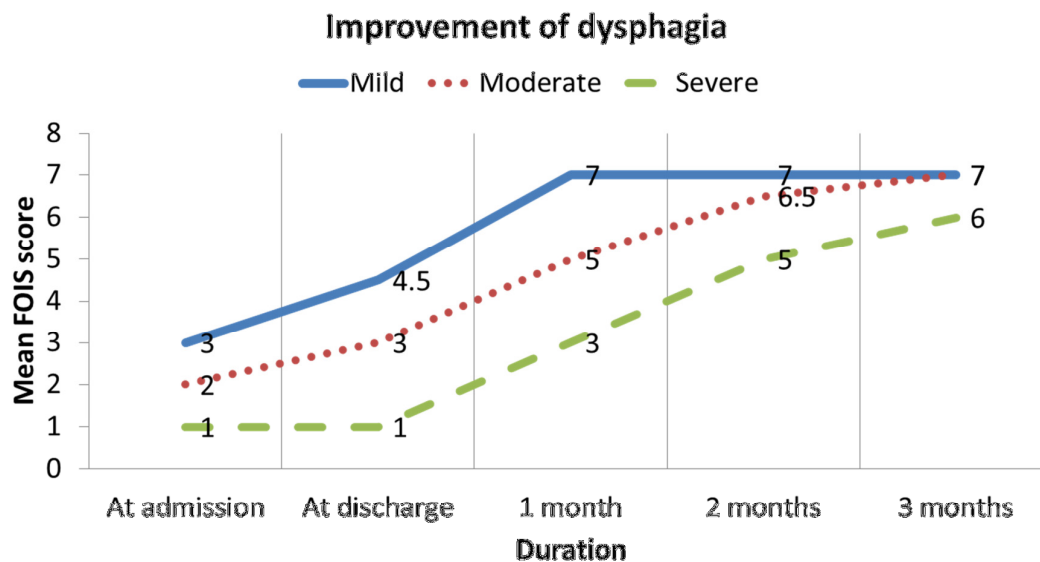


Figure 20: Improvement of dysphagia with time

The rate of pneumonia between the two groups was significantly different with 35.5% incident rate in the group with dysphagia and 4.5% in the group without dysphagia. (Figure 21)

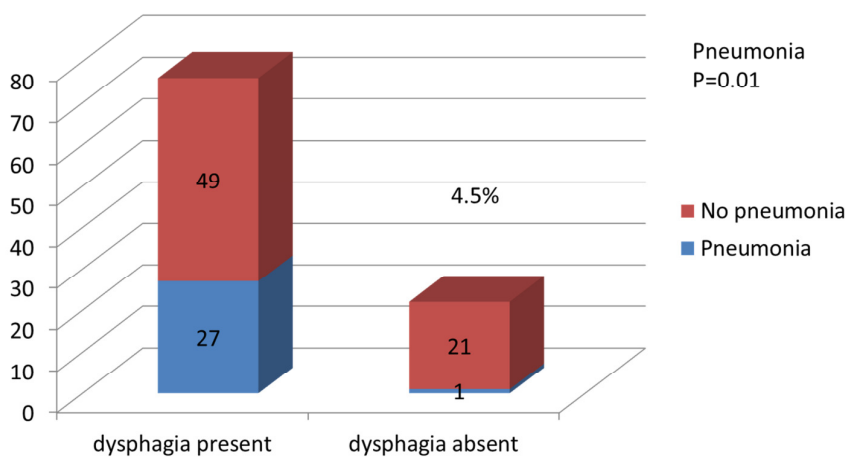


Figure 21: Incidence rates of pneumonia against the presence or absence of dysphagia

There was a significant association between the severity of dysphagia and the development of pneumonia with high incident rates of pneumonia in more severe groups of dysphagia. (Figure 22)

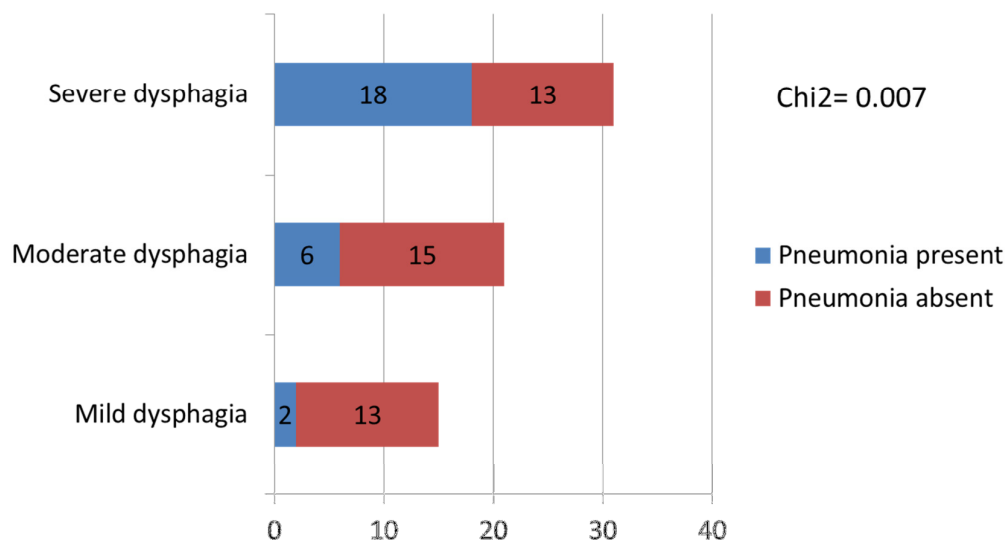


Figure 22: Relation between severity of dysphagia and pneumonia

The mortality rate was significantly different between the two groups with a 28% mortality rate in the group having dysphagia and 4.5% mortality in the group without dysphagia. (Figure 23) There was no significant difference in the day of death in between the two groups. The median day of death in the group with dysphagia was 20.8 where it was 30 in the group without dysphagia. The p value was 0.429.

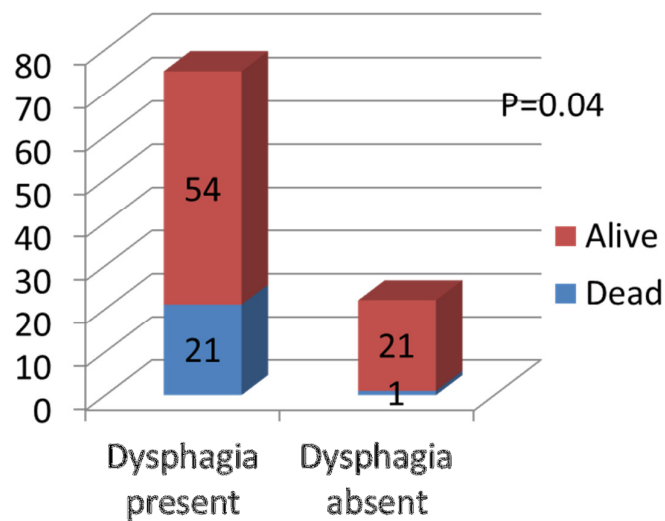


Figure 23: Relation between dysphagia and mortality

Patients with dysphagia had a prolonged hospital stay, though not statistically significant. (Figure 24)

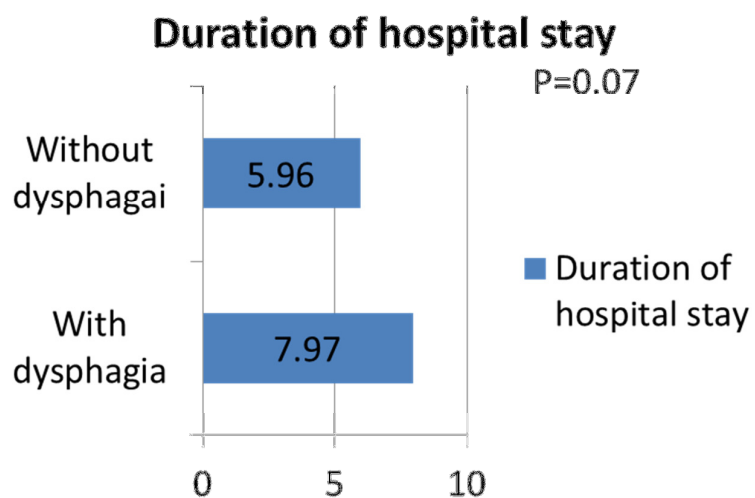


Figure 24: Relation between dysphagia and duration of hospital stay

### 5. Pneumonia:

During the follow up period, 28 patients developed pneumonia. Two patients were lost to follow up. (Figure 25)

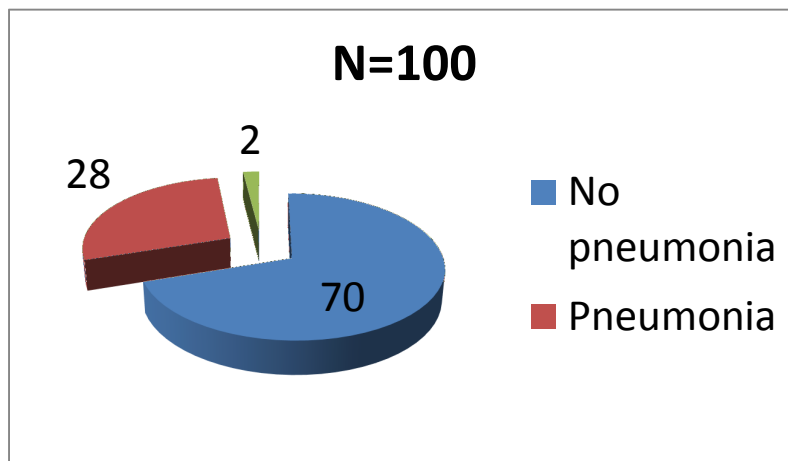


Figure 25: Incidence of pneumonia

The median day of development of pneumonia was day 5 with an interquartile range of 3 to 9.5.

### 6. Factors associated with development of pneumonia:

The prevalence of dysphagia was more in patients having pneumonia compared to those without pneumonia with a likelihood ratio of 0.004. (Figure 26)

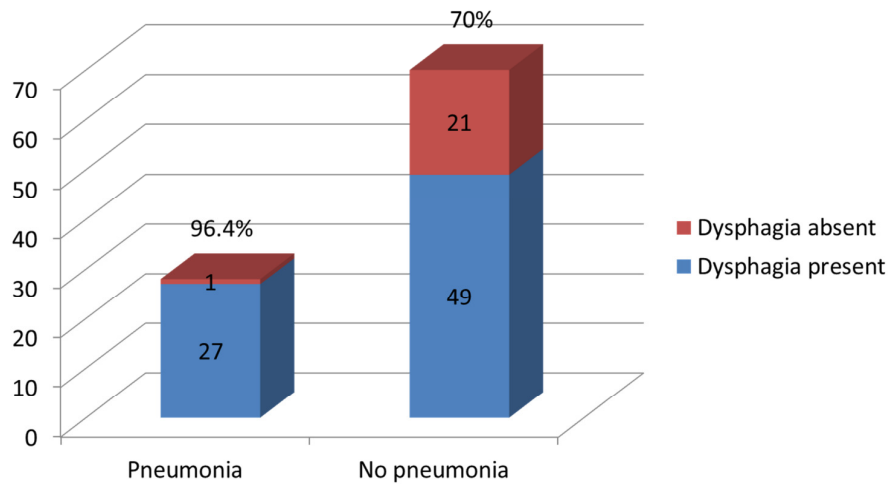


Figure 26: Relation between pneumonia and dysphagia

There was no significant difference in the sex distribution between the groups. The prevalence of diabetes and hypertension was not significantly different between the two groups. Smoking and alcohol were seen in a few patients and the distribution was not significantly different. (Table 14)

Variable	With pneumonia(28)	Without pneumonia(n=70)	P value
Sex			0.349
Males(n=66)	16	48	
Females(n=34)	12	22	
Diabetes Mellitus	13 (46.4%)	36(51.4%)	0.823
Hypertension	20(71.4%)	47(67.1%)	0.811
Smoking	3(10.7%)	7(10%)	1.000
Alcohol	2(7.1%)	8(11.4%)	0.720

Table 14: Association of sex and risk factors of stroke with pneumonia

There was no difference between the incidence of pneumonia in haemorrhagic or ischaemic strokes. There was association of pneumonia with severity of pneumonia.(Table 15)

	With pneumonia(n=28)	Without pneumonia(n=70)	P value
Type of stroke			0.081
Ischemic	16(57.1%)	54(77.1%)	
Hemorrhagic	12(42.9%)	16(22.9%)	
Side			0.066
Right	7(25.9%)	33(47.8%)	
left	20(74.1%)	56(58.3%)	
Abnormal facial nerve	25(89.3%)	52(74.3%)	0.171
Severity of dysphagia			<b>0.007</b>
Mild			
Moderate	2(7.7%)	13(31.7%)	Chi2-9.86
Severe	6(23.1%)	15(36.6%)	Df-2
	18(69.2%)	13(31.7%)	

Table 15: Association of clinical features of stroke with pneumonia

There was no significant difference in the age between the two groups. The patients with pneumonia were at a higher risk of malnutrition with a mean MUST score of 0.64 compared to 0.3 without pneumonia. The patients with pneumonia also had severe stroke with a higher median NIHSS score, more depressed consciousness with a lesser GCS score and more severe disability with a higher modified Rankin score. The severity of dysphagia was more with a lower Mann's score and a lower FOIS score. The difference between the lymphocyte counts was just significantly different between the two groups. (Table 16)



	<b>With pneumonia(n=28)</b>	<b>Without pneumonia(n=70)</b>	<b>P value</b>
Age	57.96	55.66	0.335
MUST score	0.64	0.30	<b>0.044</b>
NIHSS score(median)	14(11-20)	8(5-13)	<b>0.000</b>
GCS(median)	13(10-15)	15(13-15)	<b>0.006</b>
E	4(3-4)	4	<b>0.035</b>
M	6(5-6)	6	<b>0.012</b>
V	4.5(1-5)	5	<b>0.035</b>
Modified Rankin score(median)	4(4-5)	3(2-4)	<b>0.000</b>
Mann's score(median)	131(110-150)	172(155-190)	<b>0.000</b>
FOIS score (median)	1(1-2)	3(2-6)	<b>0.000</b>
Total count	12534	10,999	0.170
Lymphocyte percentage	14.71	18.85	<b>0.051</b>

Table 16: Association of age and various scoring systems with pneumonia

On evaluation of the association of clinical findings during swallowing assessment by speech therapist and development of pneumonia, only abnormal dry swallow was associated. (Table 17)

	<b>With pneumonia(n=15)</b>	<b>Without pneumonia(n=49)</b>	<b>P value</b>
Abnormal symmetry of lips	7(46.7%)	13(26.5%)	0.203
Abnormal symmetry of tongue	4(26.7%)	8(16.3%)	0.453
Abnormal symmetry of jaw	4(26.7%)	4(8.2%)	0.079
Abnormal Strength of tongue	13(86.7%)	37(75.5%)	0.489
Weak gag reflex	9(60%)	25(51%)	0.571
Pharyngeal sensations	4(26.7%)	4(8.2%)	0.079
Abnormal cough strength	9(60%)	19(38.8%)	0.234
Abnormal dry swallow	12(80%)	18(36.7%)	<b>0.007</b>

Table 17: Association of clinical findings of swallowing assessment with pneumonia

The clinical findings during swallowing assessment with thin liquids and thick liquids were not associated with development of pneumonia. (Tables 18 and 19)

	With pneumonia(n=5)	Without pneumonia(n=27)	P value
Abnormal lip closure	1(20%)	1(3.7%)	0.292
Drooling	0	2(7.4%)	1.000
Abnormal mastication	0	3((11.1%)	1.000
Abnormal initiation of swallow	1(20%)	7(25.9%)	1.000
Laryngeal elevation abnormal	1(20%)	3(11.1%)	0.512
Cough after swallow	1(20%)	3(11.1%)	0.512
Change in voice quality present	1(20%)	0	0.156
Wet phonation	1(20%)	0	0.156
Voluntary throat clearance	1(20%)	0	0.156
Oral residue present	1(20%)	7(25.9%)	1.000

Table 18: Association of clinical findings of swallowing assessment using thin liquid with pneumonia

	With Pneumonia(n=8)	Without Pneumonia(n=21)	P value
Abnormal lip closure	1(12.5%)	0	0.276
Drooling	1(12.5%)	1(4.8%)	0.483
Abnormal mastication	2(25%)	0	0.069
Abnormal initiation of swallow	6(75%)	9(42.9%)	0.215
Laryngeal elevation abnormal	5(62.5%)	6(28.6%)	0.197
Cough after swallow	3(37.5%)	2(9.5%)	0.112
Change in voice quality present	-		
Wet phonation	-		
Voluntary throat clearance	1(12.5%)	1(4.8%)	0.483
Oral residue present	6(75%)	11(52.4%)	0.408

Table 19: Association of clinical findings of swallowing assessment using thick liquid with pneumonia

The presence of pooling of secretions in the hypopharynx and vocal cord palsy were significantly associated with the development of pneumonia in Fibreoptic endoscopic evaluation of swallowing. (Table 20)

	With pneumonia(n=3)	Without pneumonia(n=18)	P value
Abnormal velopharyngeal closure	1(33.3%)	2(11.1%)	0.386
Pooling of secretions in hypopharynx	3(100%)	5(27.8%)	0.042
Abnormal base of tongue	0		
Medialisation of pharyngeal wall present	1(33.3%)	3(16.7%)	0.489
Vocal cord palsy			Chi2-12.5, df-2,p-
Unilateral	0	5(27.8%)	0.002
Bilateral	2(66.6%)	1(5.6%)	LR-9.184
Aspiration	2(66.6%)	6(33.3%)	0.147

Table 20: Association of findings during FEES with pneumonia.

## 6b. Logistic regression for independent factors associated with pneumonia

None of the variables were significantly associated with the development of pneumonia in logistic regression analysis.(Table 21)

	Odds ratio	SE	P value(95% CI)
Mann score	1.02	0.05	0.64(0.92-1.13)
NIHSS	1.69	0.53	0.09(0.91-3.12)
mRS	0.009	0.02	0.08(0.00-1.82)
Lymphocyte percentage	1.05	0.1	0.58(0.86-1.29)
Dysphagia	0.16	0.51	0.56(0.00-83.9)
FOIS	0.48	0.40	0.39(0.09-2.49)

Table 21: Logistic regression for independent factors associated with pneumonia

### 7. Prognosis of pneumonia:

The development of pneumonia carried a mortality rate of 59.3% which was significantly higher compared to the patients without pneumonia. (Figure 27)

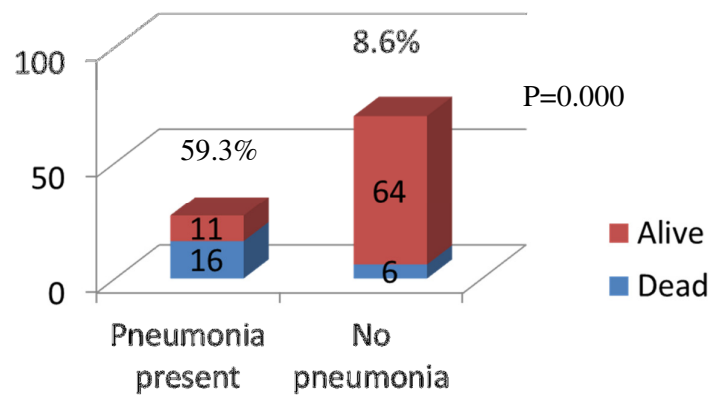


Figure 27: Relation between mortality and pneumonia

The median day of death in patients with pneumonia was day 15 which was significantly later as compared to patients without pneumonia where the median day of death was 7.5.

Development of pneumonia was associated with a significantly prolonged duration of hospitalisation with a mean hospital stay of 8.86 days in those who developed pneumonia compared to 6.33 days in those who did not develop pneumonia. (Figure 28)

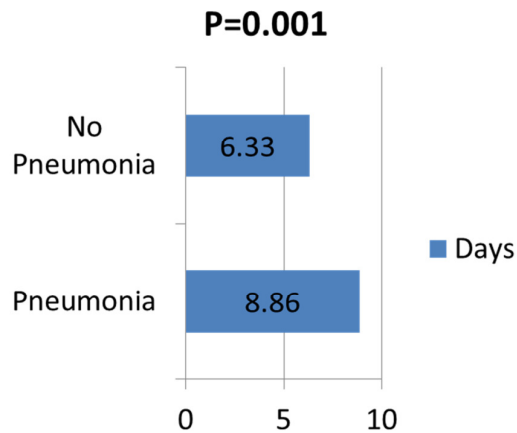


Figure 28: Relation between pneumonia and duration of hospitalisation

### 8. Utilisation of health services:

Out of 100 patients, 70 patients came for follow up. (Figure 29)

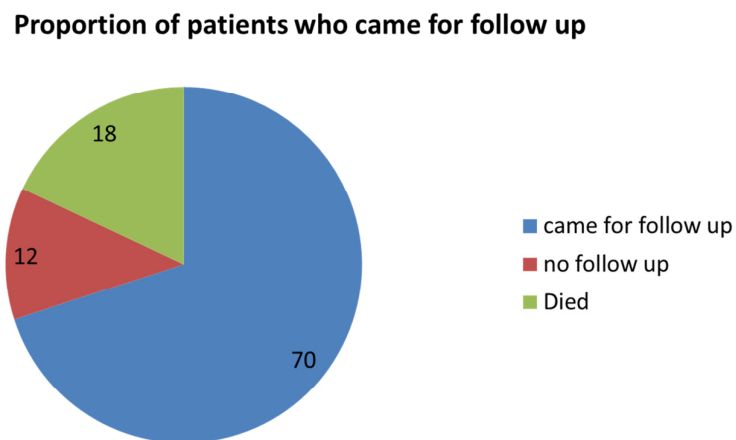


Figure 29: Proportion of patients who came for follow up

Ryle's tube was removed after assessment and under supervision of a physician in 23 patients and 29 patients had the tube removed at home. (Figure 30)

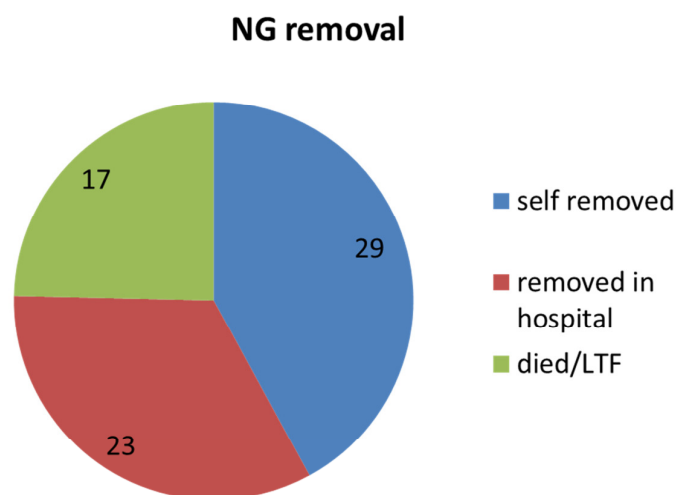


Figure 30: Location of Ryle's tube removal

## DISCUSSION

### **1. Clinical features and demographic details of stroke- similarities and differences from other studies.**

The population in this study is younger with a median age of 55 compared to most western studies with a median age of around 70.(29)(28) The sex distribution was different in different studies and our study showed male preponderance. Compared to western studies in which diabetes was seen in around 20 percent of patients with stroke (79), the prevalence of diabetes in stroke patients in our study was higher of 50 percent. The prevalence of hypertension is also higher. Smoking and alcoholism were seen in only 10 percent of patients.

Most of the strokes in this study were ischemic in anterior circulation with partial occlusion. Most of the patients had moderate severity of stroke with a median NIHSS score of 11. Most of the patients were in good consciousness with a median GCS score of 15. Majority of the patients were at low risk of malnutrition, as assessed by MUST score (median-0). Most of the patients had moderate disability with a modified Rankin score of 4. In the study conducted by Okubo et al(30) majority of the patients had NIHSS score more than 12. The majority of strokes involved middle cerebral artery territory in other studies also.(28)(31)

## **2. Diagnosis of dysphagia**

Dysphagia is a common condition after stroke. In our study, dysphagia was assessed mainly by two methods- using Mann's assessment of swallowing ability and assessment by speech therapist. Fibreoptic endoscopic evaluation of swallowing could be done only in a few patients. The incidence of dysphagia found using Mann's assessment of swallowing ability was 68 percent. This is in contrast to other studies which used the same method and found an incidence of 34 percent(46) and 51 percent(28). Another study which used modification of this method found an incidence of 36.4 percent (80). However, on assessment by speech therapist, the incidence was found to be 77 percent. Comparing with other studies which used clinical examination by speech therapist in diagnosing dysphagia, the incidence was varied with Baroni et al (31) finding an incidence of 63 percent whereas Smithard et al(29) and Okubo et al(30) found a lesser value of 44 percent and 32 percent respectively.

The overall sensitivity of Mann's assessment was found to be 82 percent in our study. Also, it was found that taking a higher cut-off value of 181, sensitivity can be improved to 100 percent. In this study, majority of patients had severe dysphagia. This is similar to study done by Baroni et al(31) in which 43 percent of the patients had severe dysphagia.

FEES has been used as gold standard in several studies and all the studies reported the presence or absence of aspiration during this procedure (81)(82)

Radhakrishnan et al (77) reported experience in using FEES to guide the decision



making for oral feeding in stroke patients and found it to be very useful. In our study, it was done in 21 patients and additional abnormal findings of vocal cord palsy and pooling of secretions were noted. Though five patients with mild to moderate dysphagia on clinical assessment were advised oral feeds, none of the patients developed pneumonia and in most of them, Ryle's tube was removed at the time of discharge. 3 patients had prolonged Ryle's tube stay of 2 to 4 weeks and two of them were found to be requiring multiple swallows while feeding during FEES.

### **3. Factors associated with dysphagia**

The factors associated with dysphagia in this study are higher NIHSS score, depressed consciousness, greater modified Rankin score. These were similar to other studies. In the study done by Baroni et al (31) showed the variables with high odds for presence of dysphagia included level of consciousness, previous stroke, sensorimotor alteration, presence of oxygen aid and higher grade on modified Rankin score. In another study (32) only depressed sensorium was associated with dysphagia. In a study conducted by Barer et al(83), depressed consciousness, presence of gaze abnormality and sensory inattention were significantly associated with dysphagia. In our study, female sex and presence of facial weakness was associated with presence of dysphagia and smoking was found to be a protective factor. However, only higher NIHSS score and weak gag reflex were the independent factors found in logistic regression.

Several clinical variables during speech therapist's assessment of swallowing were found to be significantly associated with the presence of dysphagia in univariate analysis, which are- decreased strength of lips, tongue, jaw and weak gag reflex, poor voluntary cough and abnormal dry swallow. It was also found that during assessment with thin liquids, delay in initiation of swallow and presence of oral residue were associated with dysphagia. However, none of the findings during assessment with thick liquids were associated. So, assessment of swallowing with thin liquids may be more useful compared to thick liquids. Semi solids and solids were tested in a very few patients in our study. Similar analysis was done in other studies, but the variables were analysed against presence or absence of aspiration on Videofluoroscopic Modified Barium Swallow. (84)(85)

#### **4. Prognosis of dysphagia**

Most of the patients with dysphagia improved in our study. Patients with mild dysphagia improved faster compared to moderate dysphagia and patients with moderate dysphagia improved faster than patients with severe dysphagia. There was complete recovery at the end of 2 months in patients with mild to moderate dysphagia and in patients with severe dysphagia, 9 patients (41.9%) had persistent swallowing problems and 4 were tube dependent. Similar results were found in another study, (86) in which aspiration resolved in 92% of patients at the end of 3 months. In another follow up study, dysphagia improved from 51% at admission to 27% at 7 days and at 6 months only 8% had swallowing difficulty.(87) Compared to these studies, our study assessed the improvement in swallowing ability with

regard to severity of dysphagia. But, contrast to other studies where a repeat assessment is done by speech therapist or instrumental assessment, our study assessed subjective and informant assessed swallowing ability at follow up using FOIS scale.

## **5. Incidence of pneumonia**

The incidence of pneumonia was found to be 28 percent in this study. This is in agreement with other studies in a meta-analysis in which the incidence of stroke associated pneumonia was found to vary between 3.9% and 44% in stroke units. The incidence in ICUs was much higher(69). In one study done by Sellers et al(66), the incidence of pneumonia was lower (18.9%) and in another study done by Dziewas et al(75) the incidence was 44%. The difference in incidence was related probably due to the different patient population studied. The population in the study done by Sellars et al was a mixture of varying severities of dysphagia whereas Dzeiwas et al studied patients exclusively on Ryle's tube feeding implying severe form of dysphagia.

## **6. Factors associated with pneumonia**

In this study, greater severity of dysphagia, lower MUST score, higher NIHSS score, Low GCS, higher modified Rankin score, higher Mann score, and abnormal dry swallow were associated with pneumonia. This was similar to other studies.(69)(66)(75). The other studies found additional factors like male sex, dysarthria, uncontrolled sugars, COPD and low albumin levels. Sellars et al(66) in

addition found an association between higher oral cavity score, positive bacterial cultures from oral swabs and pneumonia. Smithard et al (29) found that presence of aspiration on videofluoroscopy was not associated with development of pneumonia. Similar result was found in this study in which the presence of aspiration on endoscopic evaluation of swallowing was not associated with pneumonia. However, pooling of secretions and vocal cord palsy were associated with pneumonia.

## **LIMITATIONS**

One of the limitations of this study is that the calculated sample size was not achieved in the available time period. But, we are planning to continue the study in future.

The other limitation is that instrumental assessment of swallowing could not be done for all the patients. But, in the patients in whom it was done, there was good correlation with the clinical assessment and none of the cases of dysphagia were missed.

The follow up of improvement of swallowing was not based on objective clinical assessment, but depended on patient's and caregiver's history. The fear of such a limitation is that dysphagia and aspiration goes unnoticed causing pneumonia, but in this study, none of the patients with good FOIS scores developed pneumonia after discharge and 3 patients who developed pneumonia after discharge reported lower FOIS scores.

## **CONCLUSIONS**

1. The incidence of dysphagia is very high in acute stroke (77%) and it improves in a majority of patients (76.6%) over 3 months.
2. Weakness of gag reflex and initial severity of stroke are the major factors predicting dysphagia.
3. Dysphagia is significantly associated with pneumonia (35.5%) and high mortality (28%).
4. Pneumonia in patients with stroke has high incidence(28%) and occurs around fifth day post stroke.
5. No independent factors predicting pneumonia were identified in this study.

## **FUTURE DIRECTIONS FOR RESEARCH**

1. To study different methods of diagnosing dysphagia and to develop a bedside tool with high sensitivity and specificity to diagnose dysphagia.
2. To do a case-control study to identify factors leading to development of pneumonia.
3. To do interventional studies aiming to decrease the incidence of pneumonia in stroke.

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## **ANNEXURES**

### **PATIENT INFORMATION SHEET**

Study title: Swallowing dysfunction in stroke (SWADIS STUDY)

Study pattern: Prospective cohort study

Place of Study: Christian Medical College, Vellore

Name of the Principal Investigator: \*\*\*\*\*, PG Registrar, Department of General Medicine

Name of the Guide: Dr. Thambu David, Professor, Department of Medicine

Approximate Number of Subjects: 220

#### **Information sheet**

**Introduction:** You are invited to take part in this research study to study the swallowing dysfunction and its complications after stroke. This study aims to look at the magnitude of the problem and the complications and also to find out factors associated with development of pneumonia in stroke patients so that steps to tackle this problem can be taken in future.

**Purpose of the research:** Dysphagia is difficulty in swallowing. When a person has this difficulty, there is a high chance that food and saliva enter the lungs while trying to swallow. This leads to development of pneumonia and death in many cases. Though the problem of dysphagia in stroke is well documented, studies from India are very less regarding this topic which made us think that there is a need for detailed evaluation of the magnitude of problem and its characteristics. So, this study is planned.

**Participant selection:** You are being requested to participate/allow your relative to participate in this study as you/he/she have/has been admitted in the medical wards. The expected duration of the requested participation in this study would be 7 days from the time of admission into the ward, i.e., from the time of entering the study. In case you are discharged prior to that, we would only collect the information till the day you are admitted in hospital.

**Voluntary participation:** Your participation in this research is entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, the management and standard of care will remain the same. If you choose not to participate in this research project, you will still continue to receive the same

standards of treatment. You may change your mind later and stop participating even if you agreed earlier. This will in no way affect the care that we provide to you.

**Information on the research-Procedures & Protocol:** You will be evaluated by a specialist in swallowing problems and an endoscopy will be done in which a small camera is passed through the nose and your throat will be looked at. Apart from this we will collect some information on the disease that you suffer from, details of treatment as well as test results to correlate.

**Appropriate Alternate Procedures:** Other tests available for evaluation of swallowing problem are barium swallow. It is considered gold standard and gives good information. However, there is a risk of aspiration during this procedure and it involves radiation. Several studies have shown that endoscopy is as effective as barium swallow for evaluation of dysphagia. So, we are not going to do this procedure.

**Risks:** There is a risk of discomfort and slight bleeding through the nose during endoscopy which is not serious.

**Benefits:** The potential benefit is that these procedures including specialist assessment and endoscopy are not routinely done in patients admitted to the medical wards. The information we find in these procedures will be informed to your treating doctors regarding it.

**Reimbursements:** You will not be charged for the cost endoscopy or swallowing assessment. There are no other incentives. You will not be paid for your participation in the study.

**Confidentiality:** We will ensure confidentiality of your name and no information that identifies you will be present once we analyze the information and send it for publication.

**Sharing of the result:** Once the study is completed we will have a dissemination meeting and share results with Health care workers so that future patients may benefit from your participation

**Right to Refuse or Withdraw:** You do not have to take part in this research if you do not wish to do so. You may also withdraw participating in the research after giving the consent. It is your choice and all of your rights will be respected. The treatment will not be affected in any way.

This proposal has been reviewed and approved by the research and ethics committee of the hospital whose task it is to make sure that research participants are protected from harm.

If there are any further queries regarding this study or regarding the rights of the participants, you can contact me at

\*\*\*\*\*

PG Registrar,  
Department of General Medicine,  
Christian Medical College, Vellore.  
Ph.No.: 7373971689

@ Me/I – Principal Investigator

# You – Subject/Participant

Date:

### **INFORMED CONSENT FORM FOR SUBJECTS**

Informed Consent form to participate in a research study

Study Title: SWADIS STUDY

Study Number: \_\_\_\_\_

Subject's Initials: \_\_\_\_\_ Subject's Name:

\_\_\_\_\_

Date of Birth / Age: \_\_\_\_\_

(Subject)

(i) I confirm that I have read and understood the information sheet dated \_\_\_\_\_ for the above study and have had the opportunity to ask questions.

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published.



(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

(v) I agree to take part in the above study.

(vi) I am aware of the Audio-visual recording of the Informed Consent.  
(Click here for Audio Visual guidelines)

Signature (or Thumb impression) of the Subject/Legally Acceptable

Date: \_\_\_\_/\_\_\_\_/\_\_\_\_

Signatory's Name: \_\_\_\_\_

Signature:

Or

Representative: \_\_\_\_\_

Date: \_\_\_\_/\_\_\_\_/\_\_\_\_

Signatory's Name: \_\_\_\_\_

Signature of the Investigator: \_\_\_\_\_

Date: \_\_\_\_/\_\_\_\_/\_\_\_\_

Study Investigator's Name: \_\_\_\_\_

Signature or thumb impression of the Witness: \_\_\_\_\_

Date: \_\_\_\_/\_\_\_\_/\_\_\_\_

Name & Address of the Witness: \_\_\_\_\_

## CLINICAL RESEARCH FORM

Serial number :  
communication, Ph No.

Address for

Hospital number :

Date of admission:

Date of discharge :

Duration of hospital stay:

Duration between stroke and presentation:

Age :

Sex :

(Circle the appropriate values)

	DIABETES	HYPERTENSIO N	SMOKING	ALCHOHO L
TYPE	THROMBOTI C	EMBOLIC	HEMORRHAGI C	
SITE	TACI	PACI	LACI	POCI
	CORTICAL	SUB CORTICAL		
FRONTA L	TEMPORAL	PARIETAL	OCCIPITAL	MULTIPLE
	RIGHT	LEFT		
		FACIAL N	12 th N	
GAG	ELEVATION OF SOFT PALATE	CONSTRICION OF PHARYNX	CLOSURE OF GLOTTIS	

MUST SCORE :

FOIS SCORE :

NIHSS SCORE:

GCS :

Modified Rankin Score :

## SWALLOWING ASSESSMENT

Dysphagia- yes/no

Mann's swallowing score :

Recommendation : Ryle's tube feeding / feeds with specific consistency / normal feeds

## ENDOSCOPIC ASSESSMENT

- 1.Velopharyngeal closure - normal/abnormal
2. Appearance of hypopharynx and larynx at rest – normal/abnormal
- 3.Base of tongue- normal/ abnormal
- 4.Pharyngeal wall medialisation – present /absent
- 5.vocal cords- normal/unilateral /bilateral
- 6.Aspiration – present/ absent
- 7.Adverse effects-bleeding-yes/no

## FOLLOW UP

- 1.Pneumonia – yes/ no
- 2.Day post stroke for pneumonia – 1/2/3/4/5/6/7/ after discharge
- 3.Death –yes/no
- 4.Is death due to aspiration pneumonia-yes/no
- 5.Day post stroke for death-
- 6.Day post stroke for Ryle's tube removal-
- 7.MUST Score at discharge -

Discharge	1 month	2 months	3 months
FOIS			



